

**STUDY ON PERSISTENT UTERINE ARTERY DIASTOLIC
NOTCH –A PREDICTOR OF HYPERTENSIVE
DISORDERS OF PREGNANCY AND FETAL GROWTH
RESTRICTION
- PROSPECTIVE STUDY**

Dissertation submitted

In partial fulfillment of the requirements for the degree of

**M.D BRANCH II
OBSTETRICS AND GYNAECOLOGY**



**Kilpauk Medical College
The Tamilnadu Dr. M.G.R. Medical University
Chennai, Tamilnadu
March 2010**

CERTIFICATE

This is to certify that the dissertation entitled “**STUDY ON PERSISTENT UTERINE ARTERY DIASTOLIC NOTCH –A PREDICTOR OF HYPERTENSIVE DISORDERS OF PREGNANCY AND FETAL GROWTH RESTRICTION- PROSPECTIVE STUDY**” is the bonafide original work of **Dr. P. UMA RANI** under the guidance of **Dr. H.K. Fathima MD., DGO.,** Head of Department of Obstetrics and Gynecology, KMCH Chennai in partial fulfillment of the requirements for MD (Obs and Gyne) branch II examination of the Tamil Nadu Dr. M.G.R Medical university to be held in March 2010. The period of postgraduate study and training was from May 2007 to March 2010.

Prof. DR. H.K.FATHIMA MD., DGO.,
Professor and Head
Department of Obstetric and Gynaecology,
Kilpauk Medical College and Hospital
Chennai -600 010

Prof. DR.KANAGASABAI MD.,
THE DEAN
Kilpauk Medical College and Hospital
Chennai – 600 010

ACKNOWLEDGEMENT

I am extremely thankful to **Prof. Dr.V.Kanagasabai, M.D., Dean,** Government Kilpauk Medical College and Hospital, Chennai. who has granted permission to do this study in this institution.

I take this opportunity to express my deepest sense of gratitude to **Prof Dr. H.K.Fathima M.D.D.G.O,** Head of the Department of Obstetrics and Gynaecology, Kilpauk Medical College, Chennai for encouraging me and rendering timely suggestions and guiding me through out the course of this study. I will be forever indebted to her for her constant support.

I sincerely thank my **Prof. Dr. Famidha M.D.D.G.O,** **Dr.Meenalochini, M.D.D.G.O,** **Dr.Rajini, M.D.D.G.O,** **Dr. Yuvarani M.D.D.G.O** and **Dr. Maheswari M.D.D.G.O** for their support and guidance.

I am extremely thankful to **Prof Dr. M. Sivalingam, MD.,** H.O.D of Radiology for providing valuable support and guiding through the study.

I am extremely thankful to all the Assistant Professors of the department of Obstetrics and Gynaecology for their guidance and support through out my study period in this institution.

I wish to thank **Mr. Padmanaban**, statistician for his useful inputs.

I wish to express my gratitude to my husband for his support throughout my study.

I also wish to express my gratitude to my friends and colleagues who have always been a source of love, support and encouragement.

Above all my sincere thanks to antenatal mothers of KMCH without whom this study would not have been possible.

LIST OF ABBREVIATIONS USED

FGR	-	Fetal Growth Restriction
HTD	-	Hypertensive Disorders of Pregnancy
USG	-	Ultrasound
HZ	-	Hertz
KHZ	-	Kilohertz
MHZ	-	Megahertz
O ₂	-	Oxygen
LBW	-	Low Birth Weight
NHBPEP	-	National High Blood pressure education programme
BP	-	Blood pressure
HT	-	Hypertension
PGF2 α	-	Prostaglandin F2 α
SD	-	Standard Deviation
SGA	-	Small for gestational age
DES	-	Diethyl stilbesterol
NST	-	Nonstress test
BPP	-	Biophysical Profile
EFW	-	Estimated Fetal Weight
FL	-	Femur length

IUD	-	Intrauterine death
SLE	-	Systemic lypus eythematosus
Wks	-	Weeks
Sensi	-	Sensitivity
Speci	-	Specificity
NPV	-	Negative Predictive Value of the test
PPV	-	Positive Predictive Value of the test
LR (+) Test	-	Likelihood Ratio for positive test
LR (-) Test	-	Likelihood Ratio for negative test
H/O	-	History Of
NICU	-	Neonatal Intensive care unit.
FP	-	percentage of false positive
FN	-	percentage of false negative.
TP	-	True Positive
TN	-	True Negative
Art	-	Artery
RI	-	Resistance index

CONTENTS

CHAPTER	TITLE	PAGE NO.
1	INTRODUCTION	1
2	AIM OF STUDY	4
3	REVIEW OF LITERATURE	5
4	MATERIALS AND METHODS	31
5	RESULTS AND ANALYSIS	34
6	DISCUSSION	65
7	SUMMARY	72
8	CONCLUSION	75
9	BIBLIOGRAPHY	
10	ANNEXURES ❖ PROFORMA ❖ MASTER CHART ❖ KEYS TO MASTER CHART	

INTRODUCTION

INTRODUCTION

Pregnancy and childbirth is a unique experience in the life of women. It is filled with varied emotions from the time of conception till the delivery of the child. Every mother has an obvious expectation and anxiety of delivering a healthy baby. Medical science over the years evolved several investigative & monitoring mechanisms to monitor the growth of the fetus and to ensure its well being. Quality of life for both mother and newborn has now rightly become our top priority in the field of obstetrics. It is apparent that no greater services can be provided than ensuring each new born is well born.

Hypertensive disorders complicating pregnancy are common and form one of the deadly triad along with haemorrhage & infection that contribute greatly to maternal morbidity, mortality and responsible for 20% of perinatal mortality and morbidity. According to National Centre for health statistics in 2001, gestational hypertension was identified in 3.7% of pregnancies⁵.

Fetal growth & development remains one of the most complex & fascinating biologic processes known. The fetus has been described as a perfect parasite. Under ideal conditions, sufficient amounts of maternal nutrients are provided across uteroplacental circulation that functions

efficiently to meet the demands of the growing fetus. An appropriate hormonal and endocrine milieu for both the mother & fetus enables optimal growth. If the balanced interaction between mother & fetus is disturbed it leads to fetal growth restriction. Neonates weighing below the 10th percentile for their gestational age are said to have fetal growth restriction¹. Incidence of fetal growth restriction is close to 10% of all births and contributes to increased perinatal morbidity and mortality. There is a need for an effective screening test to predict these high risk pregnancies for better surveillance and timely intervention to improve maternal and fetal outcome.

The technological boundaries for fetal assessment have been extended to the embryonic period. For example Oasin and co workers found that embryonic heart rates may be predictive of pregnancy outcome.

Uteroplacental bed perfusion increases in normal pregnancy & decreases in fetal growth restriction and hypertensive disorders of pregnancy²⁹. The alterations in the uteroplacental circulations precedes the onset of FGR and hypertensive disorders of pregnancy.

Doppler ultrasound is an innovation in fetal surveillance which would indicate the state of uteroplacental and fetoplacental blood flow from which implications about the fetal condition can be made².

Uterine artery flow velocity wave forms recorded throughout the menstrual cycle and early pregnancy are usually characterized by an early diastolic notch which indicates high resistant uterine blood flow. In normal pregnancies the early diastolic notch persists until approximately 24 weeks gestation & it is rarely recorded on placental side after 24 weeks due to conversion of high resistance uterine blood flow to low resistance flow. FGR and hypertensive disorders of pregnancy is due to defective placentation which leads to persistence of diastolic notch. Hence this study is done to predict the occurrence of these two disorders by using persistence of uterine artery diastolic notch.

AIM OF THE STUDY

AIM OF THE STUDY

To find out the correlation between persistence of uterine artery diastolic notch by doppler and development of fetal growth restriction and hypertensive disorders of pregnancy.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Doppler study

Doppler velocimetry is a noninvasive technology that uses high frequency sound waves for the investigation of blood flow. It yields a wide spectrum of hemodynamic information.

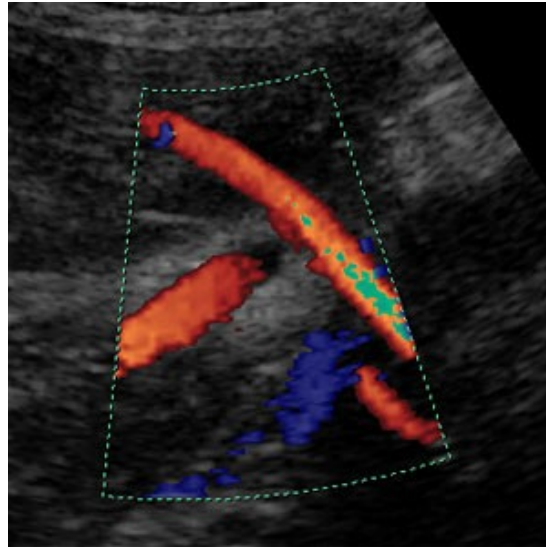
Doppler studies are based on the ‘Doppler effect’¹⁹. It is as follows,

“When a high frequency sound wave (USG) is directed toward a moving target, the reflected sound wave will have a different frequency than the emitted sound. The magnitude of this frequency shift is proportional to the velocity of the moving target from which it is reflected. When an USG beam is directed towards a blood vessel, the sound wave is mainly reflected by red blood cells which flow in it”. This is the basis for use of Doppler technology in the assessment of blood flow parameters.

Another critical event was the discovery of piezoelectric phenomenon by Pierre Curie and Jacques Curie which enabled the development of ultrasonic transducers many decades later³.

The first medical application of doppler sonography was initiated during the late 1950. Shigeo Satomura from the Institute of Scientific and

Industrial research of Osaka University in Japan developed the first Doppler ultrasound for medical Diagnostic purposes⁴.



COLOUR DOPPLER ULTRASOUND SCAN SHOWING UTERINE ARTERY

The first obstetric application of doppler USG was utilized in detection of fetal heart movements. The technique was further developed for noninvasive continuous electronic monitoring of the fetal heart rate. The first application of doppler velocimetry in obstetrics was reported by Fitzgerald & Drumm in 1977⁷.

In 1983, Teague et al⁴ developed a technique that allowed simultaneous real time imaging of fetal blood vessels & online visualisation of the doppler flow velocity - time. Other noteworthy technique have been described by Gill in 1979 & by Eiktves etal in 1980⁴ and they used a depth

specific pulsed wave doppler device with two dimensional grey scale ultrasonic imaging machine.

PHYSICAL PRINCIPLES OF DOPPLER USG³

Sound is a form of mechanical energy that travels through solid or liquid media as pressure waves. Sound waves are generated when a object vibrates in a medium. Sound waves from a vibratory source or from a reflector move across surfaces of high & low pressure. The shape of a wave form depends on the shape of the source or the interface. With doppler ultrasonics, the scattered wave form is spherical as red cells during the scattering of an incident beam.

The propagation of sound in a medium is the rate of change of position of the sound wave in unit time in that medium. It is called velocity when the direction of motion is also specified. The wave length of sound comprises of one cycle of compression and refraction. Therefore it is the distance between a pair of consecutive peaks or troughs of adjacent pressure waves. The frequency of sound is the number of such cycles occurring in one second. One cycle is hertz.

Audible sound frequency ranges from approximately 10Hz – 20 KHz. Sound with a frequency of more than 20 KHz is inaudible to the human ear & is known as ultrasonic waves. In doppler USG used for medical diagnosis,

the commonly employed frequency range is 2-10MHz. The frequency range of obstetric transducers is 2-5MHz.

To produce oscillators (or) vibrations at the rate of millions of cycles / second, special materials with piezoelectric properties are used. These piezoelectric elements are solid, non conducting substances that demonstrate physical properties whose measurement are different along different axes. When compressed in certain direction, these elements undergo electrical polarization and a corresponding voltage is generated that is proportional to the pressure conversely, when such as element is subjected to an electric field it exhibits mechanical distortion by amount proportional to the applied field. This phenomenon is known as the piezoelectric effect⁶ & allows interconnection between sound and electricity and forms the basis for the construction of doppler & other types of ultrasound transducers.

DOPPLER METHODOLOGIES

There are 3 types of doppler equipments.

1. THE CONTINUOUS WAVE DOPPLER:-

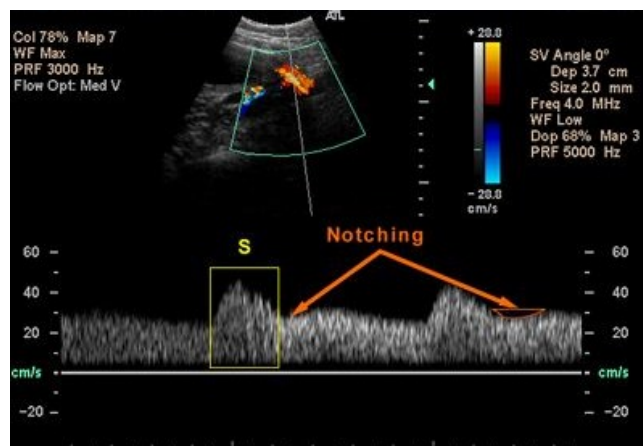
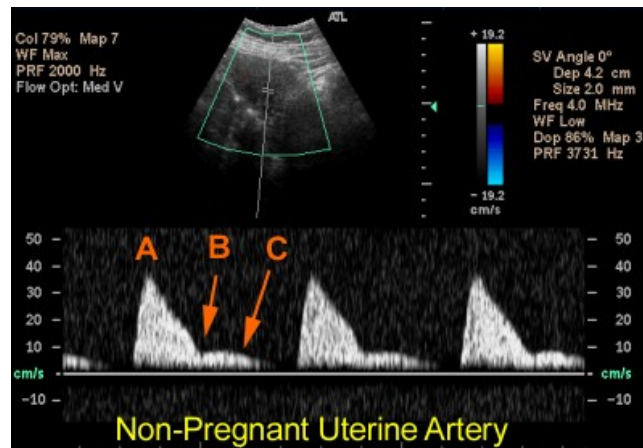
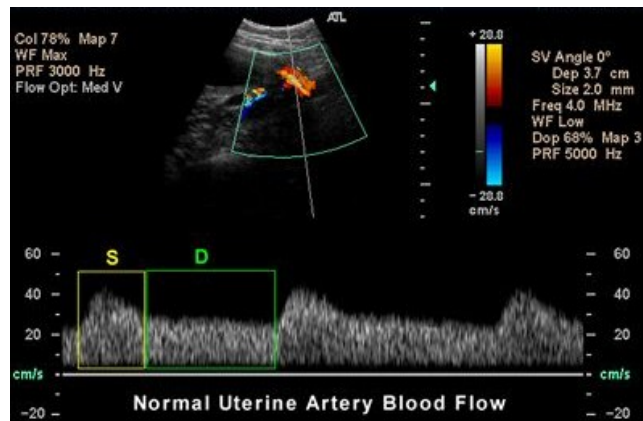
This simplest device uses 2 transducers, one continuously transmits USG waves at a fixed frequency usually within the 2-10 MHz range & the other transducer receives the waves reflected from the tissues. Most instruments use probes with 4MHz crystals.

2. THE PULSED WAVE DOPPLER:-

It contains one transducer which emits USG waves for a short period of time and then acts as a receiver for reflected waves. The transmitted beam power of the pulsed doppler is higher than that of the safety standards for fetus studies recommended by the National Institute for Health.

3. COLOUR FLOW MAPPING:-

It is based on colour encoding each pixel representing the averaged mean doppler shift. The colour is used to represent the direction, magnitude and flow characteristics of the sampled circulation. These parameters are qualitative rather than quantitative. The colour scheme is based on colour classification which is derived from the fundamental properties of light perception comprised of hue, luminance & saturation. The direction of flow in relation to the transducer is depicted in the primary colours of red (toward the transducer) & blue (away from transducer).



COLOUR DOPPLER SHOWING UTERINE ARTERY NOTCH.

DOPPLER WAVE FORM ANALYSIS OF THE FETOPLACENTAL AND UTEROPLACENTAL CIRCULATIONS:-

A recent development in fetomaternal medicine is the ability to assess the fetoplacental & uteroplacental circulations using doppler USG. The potential application of this method for the evaluation & management of certain complications of pregnancy such as fetal growth restriction & hypertensive diseases is the subject of intense investigation at the present time.^{16,17}

The doppler shift is submitted to spectrographic analysis & represented graphically as a wave form. These wave forms represent changes in the velocity of the blood flowing through the vessels. A doppler wave form is therefore shaped by hemodynamic phenomena both upstream and downstream from the measurement location. The velocity will be greater in systole and less in diastole. Analysis of the waveforms provides a qualitative measurement of the resistance to flow in the vessels that are being examined.

Most doppler indices are ratios based on the peak systolic, end diastolic & temporal average values of the maximum frequency shift. Most indices reflect pulsatility of the waveform.

The most commonly studied ratios in doppler flow are the following,

FOR ARTERIAL FLOW

$S/D = \text{Systolic peak velocity} / \text{End Diastolic velocity}$

$S - D / S = \text{resistance index}$

$S-D / \text{Mean frequency shift} = \text{pulsatility index}$

FOR VENOUS FLOW

$\text{Preload index} = \frac{\text{peak velocity during atrial contraction}}{\text{Systolic peak velocity}}$

$\text{Pulsatility index veins} = \frac{\text{Systolic diastolic peak velocity}}{\text{Time average maximum velocity}}$

$\% \text{ reverse flow} = \frac{\text{Systolic time averaged velocity}}{\text{Diastolic time averaged velocity}} \times 100$

The study of fetal vascular anatomy includes:-

A. ARTERIES

Uterine artery

Umbilical artery

Middle cerebral artery

Descending thoracic aorta

B. VEINS

Ductus venosus

Inferior venacava

Umbilical vein

In this study, persistence of uterine artery diastolic notch is used as a predictor of hypertensive disorder of pregnancy and fetal growth restriction.

Deutinger et al believed. retention of early diastolic notch is thought to represent persistence of the inherent total impedance of the uterine artery circulation.

Uterine Artery Diastolic Notch And Defective Placentation:-

Development of the uteroplacental circulation is crucial for a normal pregnancy outcome. Alteration in its development can be associated with hypertensive disorders during pregnancy and impaired delivery of O₂ & nutrients to the fetus resulting in subnormal growth.

The mean averaged S/D ratio for each trimester were

First Trimester - 5.5.

Second Trimester - 2.9

Third Trimester - 2.1

Abnormal uterine artery wave forms are those with,

1. Persistence of early diastolic notch
2. S/D ratio > 2.8 (average of Right and Left uterine artery)

It has been suggested that the presence of notch is significantly better predictor of poor pregnancy outcome than the systolic / diastolic ratio or the resistance index (RI) ^{11,12}

Thaler et al²² reported that presence of early diastolic notch is significantly better predictor of poor predictor outcome than the S/D ratio. The best sensitivities of the uterine artery waveforms are for the pregnancies with the worse outcomes such as hypertensive disorder/FGR

Uterine artery Doppler velocimetry first reported by Campbell & co workers in 1983.^{30,5} They showed that compared to pregnancies with normal uterine artery wave forms, pregnancies with abnormal wave forms were associated with more proteinuric hypertension, required more antihypertensive therapy, resulting in low birth weight and preterm delivery. Ramsay and Donner (1980) presented a summary of their anatomical studies of the uteroplacental vasculature. The first wave occurs before 12 weeks post fertilization and consists of invasion and modification of the spiral arteries of the decidua, reaching its border with the myometrium. Between 12-16 weeks post fertilization, the second wave occurs. This involves invasion of the intramyometrial parts of the spiral arteries, converting narrow lumen, muscular spiral arteries into dilated, low resistance utero placental vessels and decreased responsiveness to pressor substances. A lack of endovascular infiltration by trophoblasts into the myometrial portion of the placental bed spiral arteries results in persistence of high resistance flow and early diastolic notch.^{23,27} Defective trophoblastic invasion is the consistent finding in hypertensive disorder/ FGR.⁶

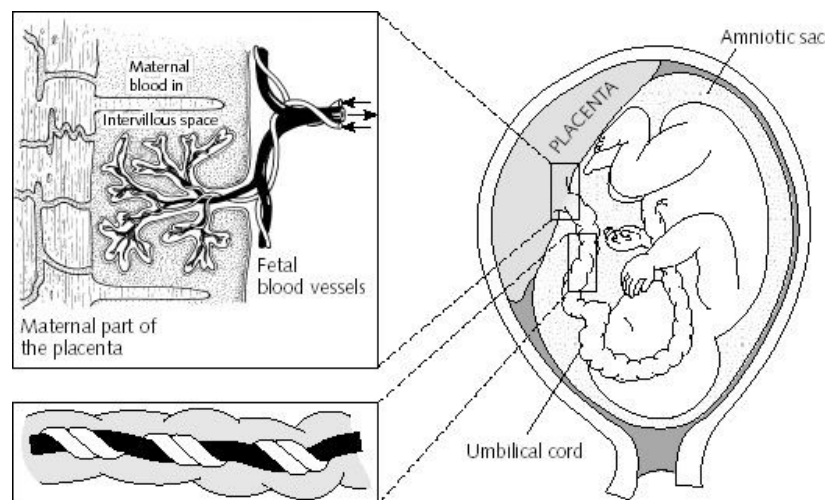
Another placental bed lesion seen in preeclampsia is an acute arteriopathy termed as acute atherosclerosis (Hertig, 1945)⁵. Here the wall of the spiral artery shows fibrinoid necrosis with lipophages & there is a mononuclear cellular infiltrate around the artery. This lesion is seen in the decidual & myometrial segments of the placental bed spiral arteries that have not undergone physiologic changes.

In normotensive pregnancies resulting in FGR, acute atherosclerosis has been seen in the decidual spiral arteries retaining their muscular coats. This finding implies that these lesions are not particular to preeclampsia.²⁶

To evaluate uterine artery doppler wave forms, color flow mapping is used to locate the uterine arteries as they cross from medial to the iliac arteries. The doppler gate is placed within the straight portion of uterine artery before it enters the myometrium⁸.

In 1983, a study by Campbell et al was the first to demonstrate a correlation between pregnancies complicated by hypertension and growth restrictions and end diastolic velocities in the arcuate arteries. This finding was associated with low birth weight, fetal distress, lower APGAR scores and increased cesarean section rate. Furthermore, proteinuria and severe hypertension correlated significantly with persistent notch.

Schulman & coworkers in 1984⁹ studied the characteristics of uterine artery velocity wave forms in nonpregnant & pregnant women. In non pregnant situation, the wave forms were typical of high resistance pattern & after ovulation, the vascular resistance decreases.



NORMAL PLACENTATION

Fleischer et al ¹⁰ in 1986 demonstrated the presence of an early diastolic notch in the uterine artery after 26 weeks gestation correlated significantly with the clinical diagnosis of preeclampsia (or) chronic hypertension super imposed preeclampsia but not with chronic hypertension alone. Notch persistence had a positive predictive value of 93%. Trudinger¹² in 1990 applied this method of uterine doppler analysis for prediction of fetal growth restriction and hypertensive disorders of pregnancy.

In Patrick F.W.¹⁵ & Neil Arnott evaluated the clinical usefulness of doppler analysis of uterine artery velocimetry wave form in the prediction of FGR, hypertensive disorder, perinatal death & concluded that uterine arter doppler had limited diagnostic accuracy in predicting FGR, hypertensive disorder, perinatal death.

HYPERTENSIVE DISORDERS IN PREGNANCY

The classification of hypertensive disorders complicating pregnancy adopted by working group of the NHBPEP (2000) consists of 5 types as follows,

1. Gestational hypertension (formerly called as pregnancy induced hypertension)
2. Preeclampsia
3. Eclampsia
4. Preeclampsia superimposed on chronic hypertension
5. Chronic hypertension

HYPERTENSION:-

According to NHBPEP, it is defined as systolic B.P. of more than or on equal to 140 mm Hg & diastolic B.P. of more than or equal to 90 mm Hg, taken at 2 occasions 6 hours apart. Diastolic BP is determined as the disappearance of korotkoff sound (phase V).

GESTATIONAL HYPERTENSION

Hypertension without proteinuria developing after 20 weeks of gestation, during labour, puerperium in a previously normotensive, non proteinuric women and B.P returns to normal by 12 weeks postpartum.

PREECLAMPSIA:-

Hypertension associated with proteinuria $>0.3\text{g/l}$ in 24 hour urine collection or 1+ by qualitative urine examination after 20weeks of gestation, during labour, puerperium in a previously normotensive non proteinuric women and returns to normal by 12 weeks postpartum.

ECLAMPSIA:-

Convulsions / coma occurring in a patient with preeclampsia are known as eclampsia.

CHRONIC HYPERTENSION:-

It is defined as hypertension present before the 20th weeks of pregnancy (or) present before pregnancy.

CHRONIC HT SUPERIMPOSED PREECLAMPSIA:-

It is defined as proteinuria developing for first time during pregnancy in a woman with known chronic hypertension after 20 weeks of gestation.

INCIDENCE¹:-

Incidence of Hypertensive disorders of pregnancy is about 12% -22% of all pregnancies.

Preeclampsia – 5% - 8%.

RISK FACTORS:-

Nulliparous women

Multiple pregnancies

Genetic predisposition

Molar pregnancy

Environment factors

Diabetes

Obesity

ETIOLOGY⁵:-

Preeclampsia is more likely to develop in women who

- Are exposed to chorionic villi for the first time
- Are exposed to super abundance of chorionic villi as with twins (or) hydatidiform mole.
- Have preexisting vascular disease
- Are genetically predisposed to HT developing during pregnancy.

A fetus is not a requisite for preeclampsia. According to SIBAI (2003) currently plausible potential causes include,

1. Abnormal trophoblastic invasion of uterine vessels.
2. Immunological intolerance between maternal and fetoplacental tissues

3. Maternal maladaptations to cardiovascular (or) inflammatory changes of normal pregnancy.
4. Nutritional factors – Zinc, calcium, Magnesium deficiencies
5. Genetic factors

Failure of second wave trophoblastic invasion leads to diminished uteroplacental blood flow, increased pressor responses due to alteration in $\text{PGF}_2 \alpha$ and thromboxane A_2 synthesis, decreased Nitric oxide synthesis, increased endothelin -1 production, endothelial cell injury and vasospasm forms the basis for all the complications.

On the basis of placental bed biopsies by Dixon & Robertson (1961), Brosens et al (1972) & Robertson (1976)²⁹ reported that in preeclampsia, there is failure in the second wave of trophoblastic invasions. Musculo elastic media of the spiral arteries are retained & the vessel fails to dilate & remains responsive to vaso pressor agents. In preeclampsia, there is increased total peripheral resistance and decreased cardiac output. Hemoconcentration is the hallmark of preeclampsia even more in eclampsia

COMPLICATIONS OF PREECLAMPSIA:-

- Immediate – Maternal, Fetal
- Remote

IMMEDIATE:-

1. Maternal:-

- ❖ Eclampsia
- ❖ Accidental Haemorrhage
- ❖ Oliguria and anuria
- ❖ Visual disturbances
- ❖ Preterm labour
- ❖ HELLP syndrome
- ❖ Coagulation failure
- ❖ Postpartum haemorrhage
- ❖ Sepsis

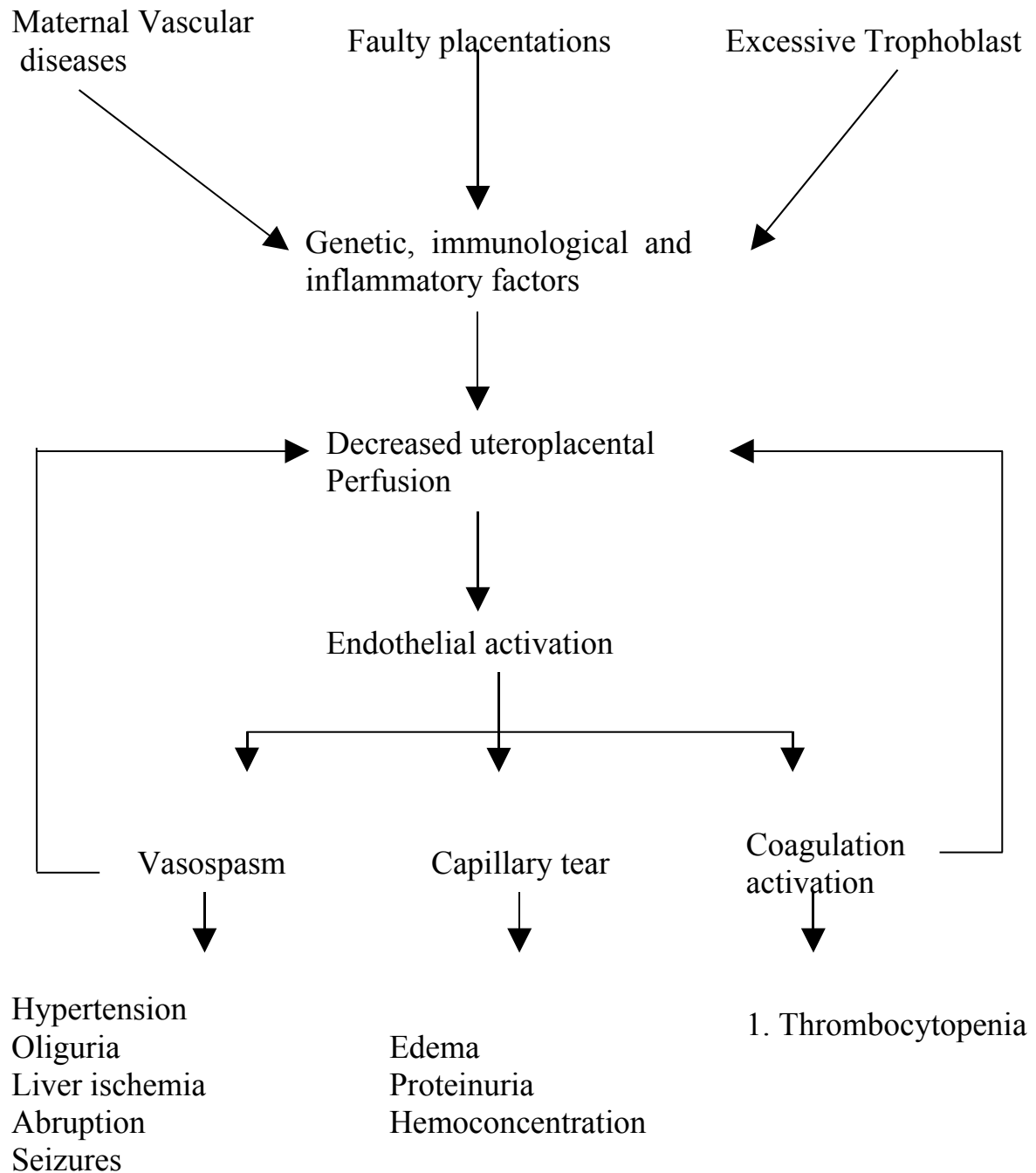
FETAL:-

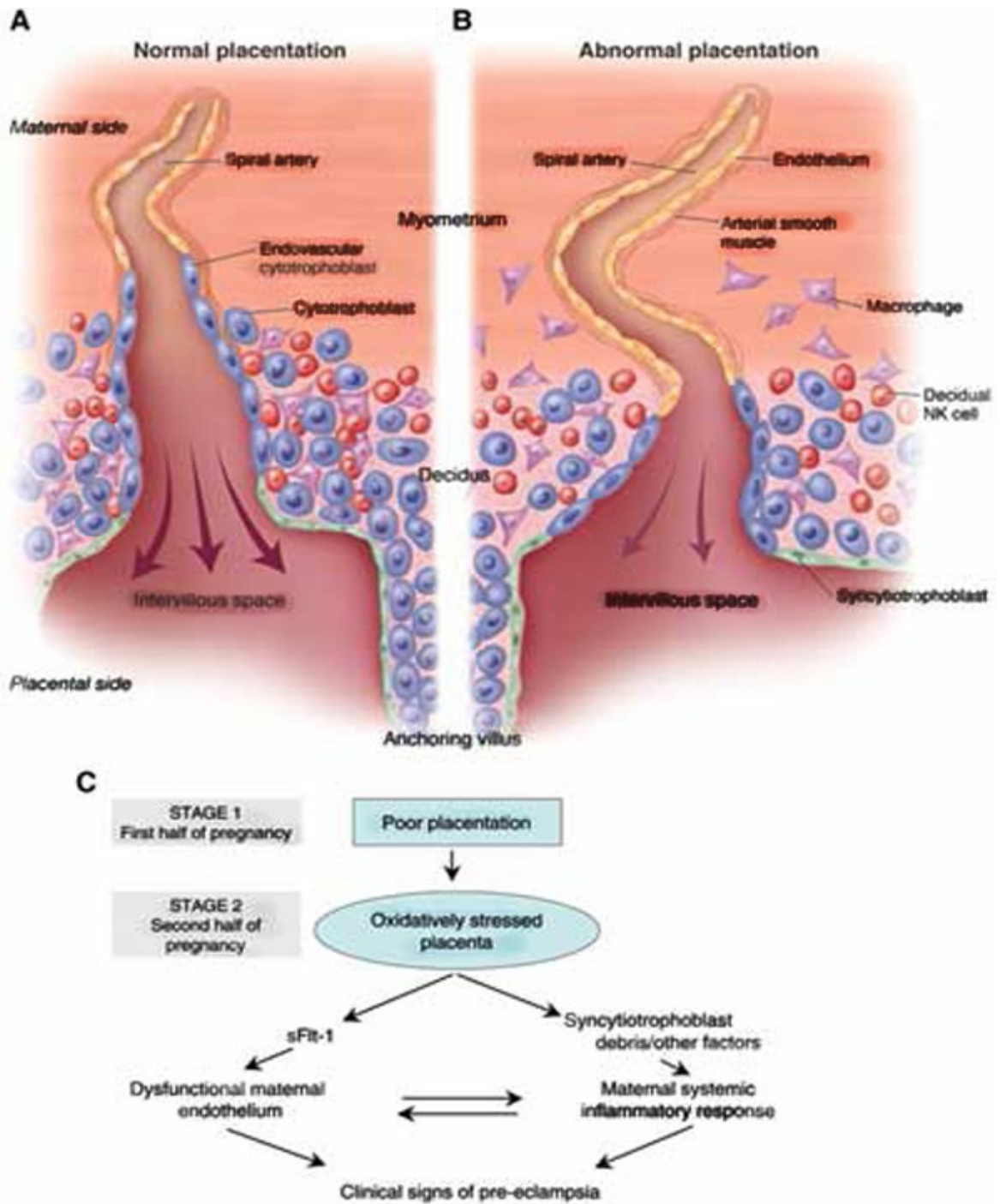
- ❖ Intrauterine death
- ❖ Fetal growth restriction
- ❖ Asphyxia
- ❖ Prematurity

REMOTE:-

- ❖ Residual hypertension -50%
- ❖ Recurrent preeclampsia – 30%
- ❖ Chronic renal disease.

PATHOPHYSIOLOGY⁵





PREDICTORS OF PREECLAMPSIA:-

Several investigators have attempted to identify early markers of defective placentation, endothelial dysfunction. Few are listed below,

1. Rollover Test
2. Serum uric acid
3. Serum. Fibronectin
4. Tests for oxidative stress
5. Serum placental peptides
6. Uterine artery doppler velocimetry- better tests among the currently available tests.¹⁰

MANAGEMENT:-

Objectives are

1. Treatment for hypertension
2. Prevention of complications
3. Identification and treatment of complications
4. Delivery of healthy baby with minimum maternal morbidity

FETAL GROWTH RESTRICTION

Neonates weighing below the 10th percentile for their gestational age or 2.SD below the mean for that gestational age are considered growth restricted¹.

It is a pathological restriction of fetal growth which occurs throughout the pregnancy both in the size of the fetus & in the functions of its various organ systems.

INCIDENCE:-

Developed countries – 4-8%

Developing countries – 7-12%

SGA– Babies with birth weight <10th Percentile for their gestational age without a pathologic restriction in their growth.

TYPES OF FGR:-

Growth restriction is the result of numerous pathologies that reduce fetal cell size. When it is early & severe, it causes a reduction in cell number.

FGR CAN BE CLASSIFIED INTO¹

Type I FGR:-

It represents fetuses that are symmetrically small & have normal head to abdomen & femur to abdomen ratios. It is otherwise known as intrinsic FGR and arises from conditions within the fetal compartment itself.

Type II FGR:-

It represents fetuses that have an abdominal circumference that is smaller than the head circumference & the femur length. This is known as extrinsic FGR & constitutes about 80% of FGR. It is due to restriction in nutrient supply due to uteroplacental insufficiency.

CAUSES OF FGR^{1,18}

MATERNAL:-

- | | |
|--|---|
| 1. Preeclampsia, Chronic hypertension. | 6. Sick cell anaemia |
| 2. Renal disease | 7. Malnutrition & malabsorption |
| 3. Connective tissue disorders | 8. Fever |
| 4. Diabetes mellitus | 9. Addictions like smoking, alcohol |
| 5. Cardiac diseases class III/IV | 10. Drugs like DES, anticancer agents, Narcotics, anticonvulsants |

Uterine causes:-

1. Decreased uteroplacental blood flow
2. Atheromatosis

3. Arteriosclerosis of decidual spiral arteries
4. Fibromyoma
5. Mullerian anomalies like bicornuate uterus /uterus didelphus.

Fetal causes :-

1. Viral infections
2. Syphilis
3. Chromosomal abnormalities
4. Multiple pregnancy
5. Heart diseases
6. Osteogenesis imperfecta

Placental factors:-

- | | |
|---------------------|-----------------------------------|
| - Abruptio placenta | - Deciduitis |
| - Placenta praevia | - Placentitis, vasculitis |
| - Thrombosis | - Edema, chorioamnionitis |
| - Infarction | - Chorioangioma, placental cysts. |

Complications of FGR:-

Maternal:-

Maternal complications are mostly due to underlying diseases like preeclampsia, cardiac disease class III and IV, Chronic HT, renal disease,

preterm labour, cesarean delivery, Oligohydramnios is present in 80% to 90% of FGR cases.

Fetal:-

Stillbirth, hypoxia, acidosis, congenital malformations.

Neonatal:-

Hypoglycemia, hypocalcemia, hypoxia, acidosis, hypothermia, meconium aspiration syndrome, polycythemia, congenital malformations, sudden infant death syndrome.

Longterm :-

Low IQ, learning & behavioural problems, major neurologic handicaps like seizure disorders, cerebral palsy, severe mental retardation & hypertension.

Diagnosis of FGR:-

The use of risk profiles based on maternal history has poor sensitivity & specificity

Clinical screening :-

This is by maternal weight gain, symphysio fundal height, Abdominal palpation.

Ultrasonic diagnosis:-

The adequacy of fetal growth cannot be determined by a single sonographic examination without a previous estimate of gestational age. So

expected date of confinement is set by early ultrasonogram.²⁵ The following parameters are useful,

- | | |
|----------------------------|---------------------------|
| 1. Biparietal diameter | 4. Head circumference |
| 2. Femur length | 5. Estimated fetal weight |
| 3. Abdominal circumference | |

Doppler velocimetry:-

Abnormal uterine artery doppler by absent or reversal of diastolic flow has been uniquely associated with FGR. Useful in management of FGR as a possible adjunct to NST and BPP.

Biometric Ratios ^{13,14}:-

Head to abdomen ratio (HC/AC):-

This ratio compares the best preserved organ (liver) with the most affected one (brain). This ratio is normal in symmetric FGR. It decreases with gestational age. Normal HC/AC ratio is greater than one upto 34-36 wks of gestation, the value is less or equal to one from 36 wks of gestation to delivery¹³

Femur to abdomen ratio (FC/AC):-

It is a gestational age independent ratio. It remains constant after 20wks at 22 ± 2 . High femur length to abdominal circumference ratio suggests fetal growth restriction.

Fetal ponderal index

$$PI = \frac{EFW}{(FL)^3} \times 100$$

At 28wks, fetal ponderal index is 1.5. It increases by 0.2 every 4 wks to reach a maximum value of 2.4 at 40 weeks. It is a gestational age independent ratio & has a constant value through out the second half of pregnancy.

Management:-

1. To confirm the diagnosis
2. To exclude anomalies
3. To treat the cause if found
4. Fetal surveillance
5. Timely delivery

MATERIALS AND METHODS

MATERIALS AND METHODS

This prospective study done to find out the correlation between persistence, of uterine artery diastolic notch and development of hypertensive disorders of pregnancy, and fetal growth restriction was undertaken in Obstetrics and Gynaecology Department at Government Kilpauk Medical College Hospital from October 2007 to June 2009. All the cases were belonged to class four and class five socioeconomic status.

Selection of Cases:-

The antenatal mothers were clinically evaluated at the Antenatal OP Department & were allocated into two groups as follows,

Group I: 100 antenatal mothers at 16-28 weeks primi/multi

Group II: 100 antenatal mothers at 16-28 weeks with previous history of FGR / IUD / hypertensive disorders of pregnancy

Inclusion criteria:-

For Group I:-

Antenatal mothers primi /multi without any previous H/O FGR / Hypertensive disorders of pregnancy /IUD

For Group II:-

Antenatal mothers with previous H/O FGR /IUD/ Hypertensive disorders of pregnancy

Exclusion criteria:-

- 1) Women with medical disorders complicating pregnancy like diabetes, cardiac diseases, chronic hypertension, SLE complicating pregnancy, chronic renal disease.
- 2) Multiple gestation.
- 3) Women with congenitally malformed fetus.

Method of study:-

AN mothers were registered at 16 wks for basic evaluation.

For all mothers, a thorough general, obstetric history was elicited and a complete general, obstetric examination was done. All the basic investigations were done. Uterine artery doppler was done between 24 & 28 weeks of gestation. Doppler characteristics evaluated for predicting FGR/ hypertensive disorders of pregnancy was persistence of uterine artery diastolic notch.

Method of Doppler study:-

The selected cases are subjected to a colour doppler which included biometry, doppler evaluation for persistence of diastolic notches in the uterine arteries of both sides. The doppler equipment consisted of a colour doppler system with a carrier frequency of 3.5MHZ. The doppler evaluation was carried out as follows. Antenatal mother is placed in a supine, slightly

left lateral position & wedge is placed under the left flank. It is important to avoid supine hypotension syndrome due to venacaval compression. For uterine artery doppler, the probe is placed 2-3cm medial to the anterior superior iliac spine. The transducer is pointed laterally and downward toward the parametrial area where the iliac vessels pierce the myometrium. The presence of diastolic notch was noted. Main outcome variables for analysis were the development of hypertension with or without proteinuria, FGR, mode of delivery, gestational age at delivery and perinatal outcome. The datas were collected & analysed statistically and evaluated critically.

RESULTS AND ANALYSIS

RESULTS AND ANALYSIS

Statistics were analysed with the computer statistical analysis system. Datas were compared using chi-Square test. Univariate and multivariate analysis of the datas were done. Since side of the uterine artery notch was not related to the outcome of interest²⁴ (HTD / FGR), so in this study it was considered as unilateral notch or bilateral notch.

The performance of the tests was evaluated by calculating sensitivity, specificity, positive and negative predictive values, likelihood ratios for abnormal and normal tests with their 95% confidence interval. The likelihood ratio is a stable predictive property of a test because it combines information from both sensitivity and specificity and is independent of prevalence.

The interpretation of likelihood ratios for positive and negative test results has been reported by Jaeschke et al.⁴⁰ A likelihood ratio of 1 indicates that the test has no predictive value for the outcome event of interest. To achieve conclusive prediction of the outcome event of interest, a likelihood ratio of more than 10 or less than 0.1 would be required for a positive and negative test results respectively. Moderate prediction can be achieved with likelihood ratios of 5-10 and 0.1-0.2, whereas likelihood ratios of 1-5 and 0.2-1 would generate only mild prediction.

In group, I, 100 cases and in group II 100 cases were selected and prospectively followed up. 3 cases in group I and 2 cases in group II were lost follow up. These cases not reported back after doppler study. The selected cases had uterine artery evaluation between 24-28 weeks gestation and followed up for development of hypertensive disorders, fetal growth restriction, gestational age at delivery, mode of delivery and perinatal outcome. Cases included were mainly belonging to class IV / class V socioeconomic status.

The following observations were made,

1. AGE DISTRIBUTION OF CASES:-

Table - I

Age	No of cases	
	Group I	Group II
18-20 yrs	27 (27.8%)	13 (13.2%)
21-30yrs	66 (68%)	76 (77.5%)
31-35yrs	4 (4.1%)	9 (9.1%)

Mean =22.1

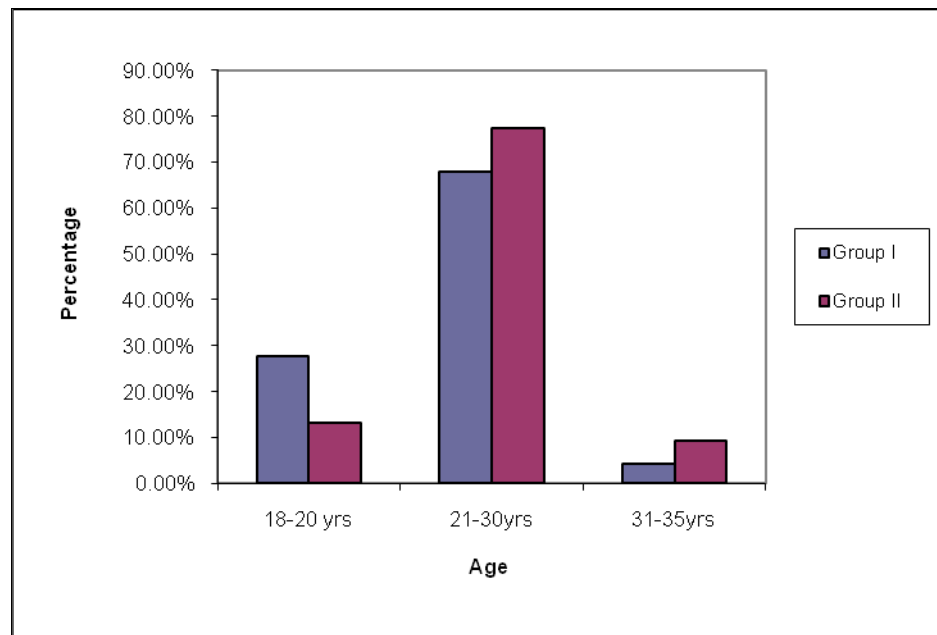
Mean =24.4

SD=2.67

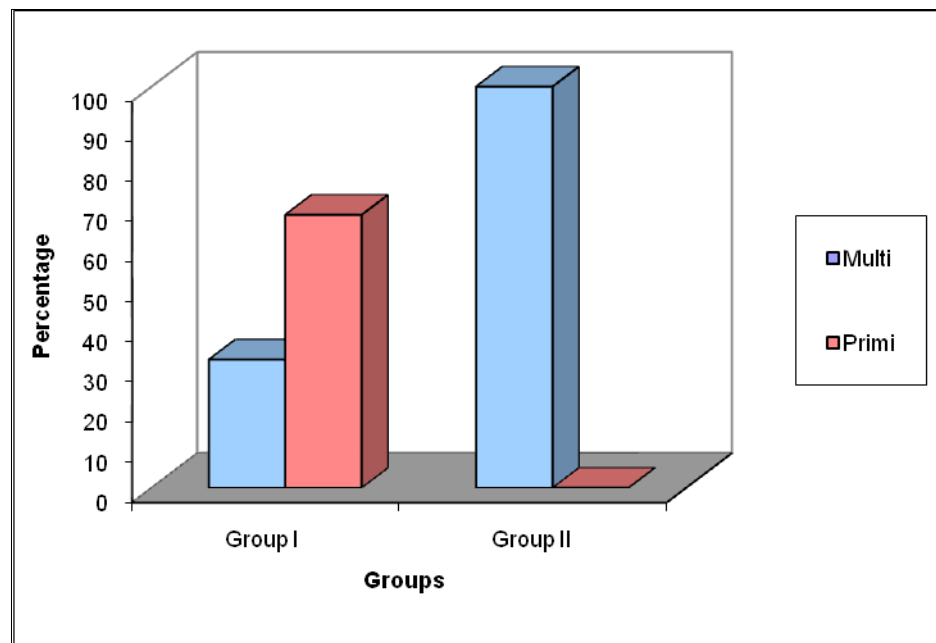
SD=3.43

In this study, 66 cases (68%) in group I and 76 cases (77.5%) in group II belonged to the age group of 21-30 yrs. 27 cases (27.8%) in group I and 13 cases (13.2%) in group II belonged to the age group of 18-20 yrs and the remaining belonged to the age group of 31- 35 years.

AGE DISTRIBUTION OF CASES



PARITY DISTRIBUTION OF CASES



2. PARITY DISTRIBUTION OF CASES :-

Table - II

Group I

Parity	Count	%
Multi	31	31.96
Primi	66	68.04
Total	97	100

In Group I, 66 cases (68.04%) were primigravida and 31 cases (31.96%) were multigravida.

Table III

Group II

Parity	Count	%
Multi	98	100
Primi	0	0
Total	98	100

In Group II, all were multigravida 98 cases (100%).

3. NOTCH DISTRIBUTION :-

Table - IV

Group - I

Notch	Count	%
Absent	75	77.32
Bilateral	11	11.34
Unilateral	11	11.34
Total	97	100

In Group I, bilateral notch was present in 11 cases (11.34%) and unilateral notch was present in 11 cases (11.34%).

Table - V

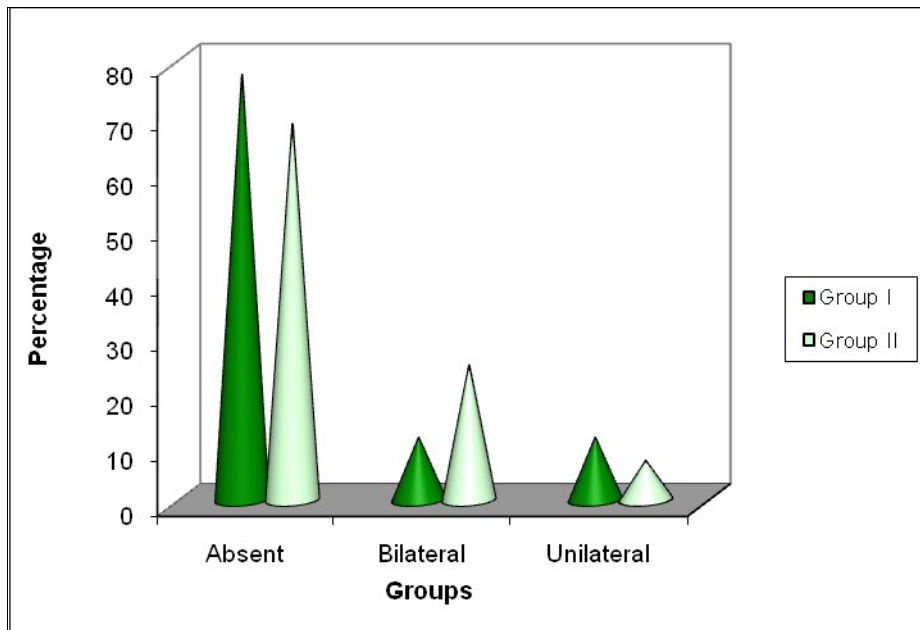
Group – II

Notch	Count	%
Absent	67	68.37
Bilateral	24	24.49
Unilateral	7	7.14

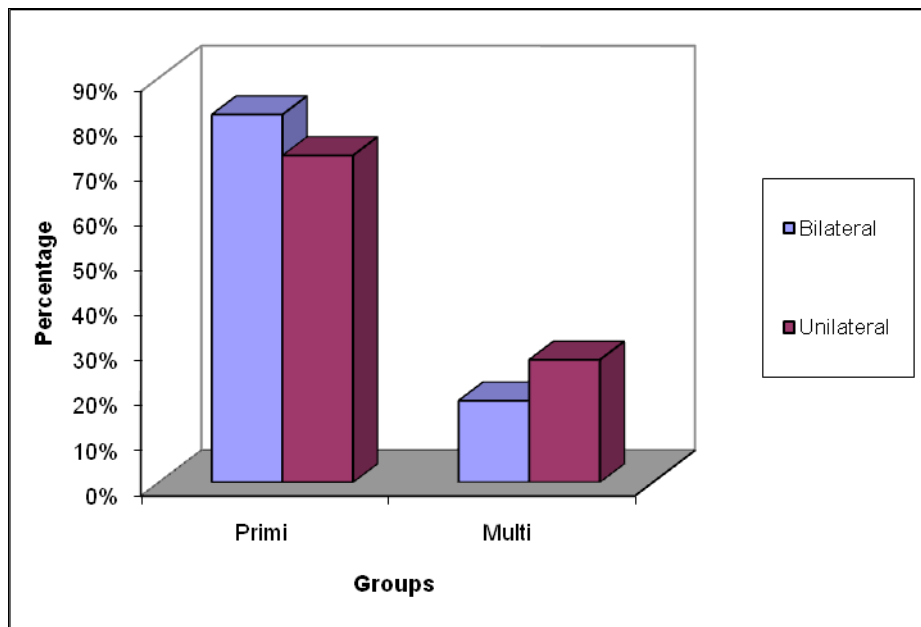
Total	98	100
--------------	-----------	------------

In Group II, bilateral notch was present in 24 cases (24.49%) and unilateral notch was present in 7 cases (7.14%).

NOTCH DISTRIBUTION



DISTRIBUTION OF CASES IN RELATION TO PARITY



4. DISTRIBUTION OF CASES IN RELATION TO PARITY:-

Table - VI

Group - I

Notch	Parity		Total
	Primi	Multi	
Bilateral	9 (81.81%)	2 (18.18%)	11
Unilateral	8 (72.72%)	3 (27.27%)	11

P = 0.022

In Group I, in cases with persistence of bilateral notch 9 cases (81.81%) were primi and 2 cases (18.18%) were multi. In cases with persistence of unilateral notch, 8 cases (72.72%) were primi and 3 cases (27.27%) were multi. P value of 0.022 indicates significant (at 5%) relationship between notch & parity. Notch is associated with primipara.

5. NOTCH AND HTD/FGR:-

Table - VII- A

Group - I

Notch	Number	Normal outcome	HTD	FGR
Bilateral	11 (11.34%)	6 (54.5%)	4 (36.36%)	3 (27.27%)
Unilateral	11 (11.34%)	7 (63.63%)	2 (18.18%)	2 (18.18%)
Total	22 (22.68%)	13 (59.09%)	6 (27.27%)	5 (22.72%)
Absent	75 (77.31%)	72 (96%)	2 (2.6%)	1 (1.3%)

In group I, 4 cases (36.36%) had HTD, 3 cases (27.27%) had FGR in the cases with persistence of bilateral uterine artery notch.

In group I, 2 cases (18.18%) had HTD, 2 cases (18.18%) had FGR in the case with persistence of unilateral uterine artery notch.

In group I, 2 cases (2.6%) had HTD, and 1 case (1.3%) had FGR in the absence of notch.

Table - VII-B

Group - I

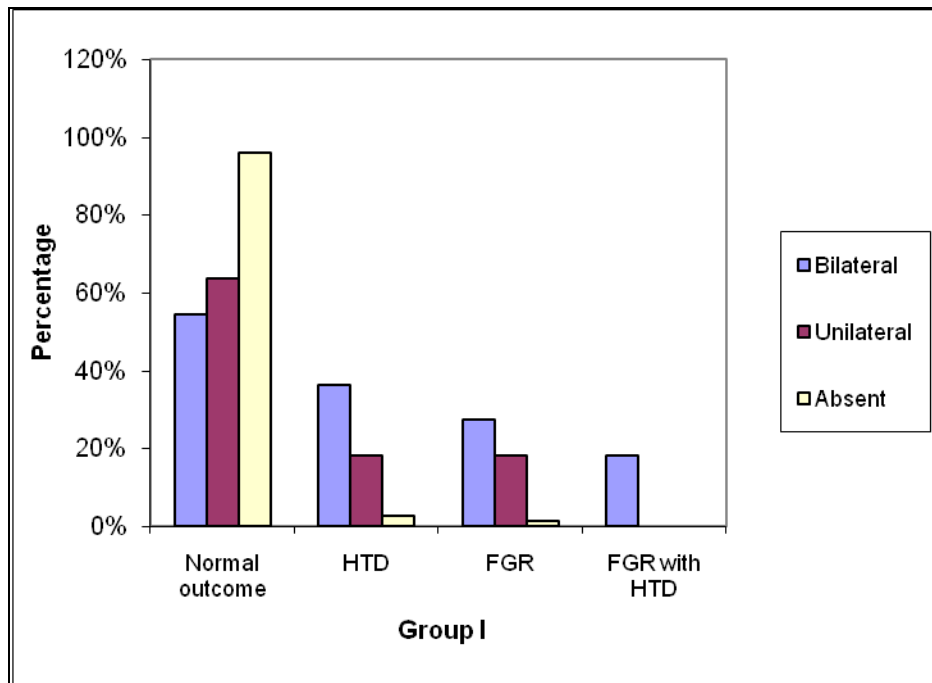
Notch	Number	FGR without HTD	FGR with HTD	HTD without FGR
Bilateral	11 (11.34%)	1 (9.09%)	2 (18.18%)	2 (18.1%)
Unilateral	11 (11.34%)	2 (18.18%)	-	2 (18.18%)
Total	22 (22.68%)	3 (13.63%)	2 (9.09%)	4 (18.18%)
Absent	75 (77.31%)	1 (1.3%)	-	2 (2.6%)

In group I, 2 cases (18.18%) had HTD with FGR in cases of persistence of bilateral uterine artery notch.

In group I, in the absence of notch and in the presence of unilateral notch, there is no occurrence of HTD with FGR.

NOTCH AND HTD/FGR

Group - I



Group – II

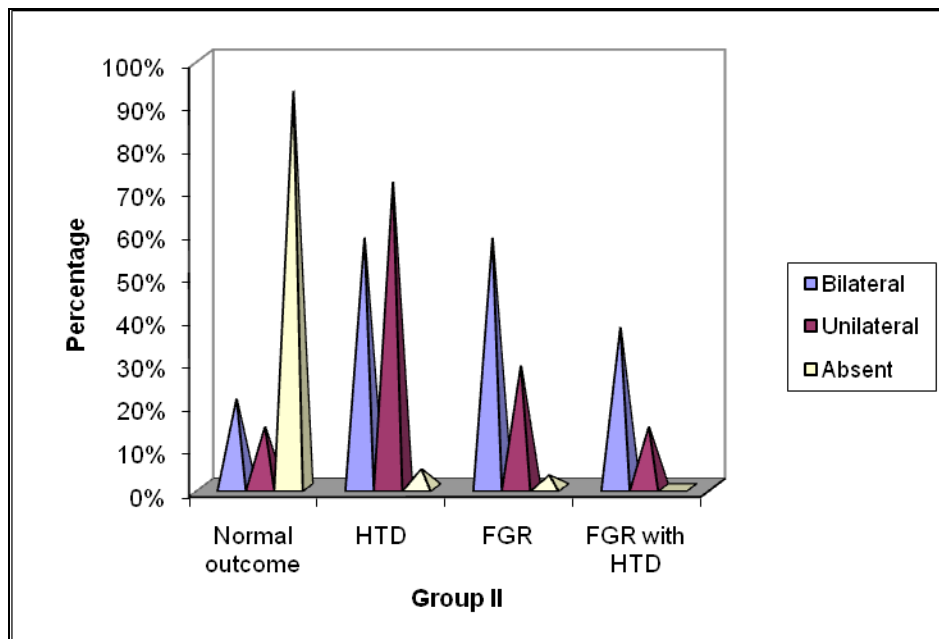


Table VIII- A

Group – II

Notch	Number	Normal outcome	HTD	FGR
Bilateral	24 (24.49%)	5 (20.8%)	14 (58.33%)	14 (58.33%)
Unilateral	7 (7.14%)	1 (14.28%)	5 (71.4%)	2 (28.57%)
Total	31 (31.6%)	6 (19.35%)	19 (61.22%)	16 (51.61%)
Absent	67 (68.36%)	62 (92.53%)	3 (4.4%)	2 (2.98%)

In group II, 14 cases (58.33%) had HTD, 14 cases had FGR in the cases of persistence of bilateral uterine artery notch.

In group II, 5 cases (71.4%) had HTD, 2 cases (28.57%) had FGR in the cases of persistence of unilateral uterine artery notch.

In the absence of notch 3 cases (4.4%) had HTD and 2 cases (2.9%) had FGR.

Table - VIII- B

Group - II

Notch	Number	FGR with HTD	FGR without HTD	HTD without FGR
Bilateral	24 (24.49%)	9 (37.5%)	5 (20.8%)	5 (20.8%)
Unilateral	7 (7.14%)	1 (14.28%)	1 (14.28%)	4 (57.14%)
Total	31 (31.6%)	10 (32.2%)	6 (19.35%)	9 (29.03)%
Absent	67 (68.36%)	-	2 (2.98%)	3 (4.4%)

In group II, 9 cases (37.5%) had HTD with FGR in cases with persistence of bilateral uterine artery notch.

In group II, 1 case (14.28%) had HTD with FGR in the presence of uterine artery notch.

Table - IX-A

OVER ALL RESULTS:-

Notch	Number	Normal outcome	HTD	FGR
Bilateral	35 (17.9%)	11 (31.42%)	18 (51.42%)	17 (48.57%)
Unilateral	18 (9.2%)	8 (44.44%)	7 (38.88%)	4 (22.22%)
Total	53 (27.17%)	19 (35.84%)	25 (47.16%)	21 (39.62%)
Absent	142 (72.8%)	134 (94.36%)	5 (3.5%)	3 (2.1%)

Table - IX-B

Notch	Number	FGR with HTD	FGR with out HTD	HTD without FGR
Bilateral	35 (17.9%)	11 (31.42%)	6 (17.14)	7 (20%)
Unilateral	18 (9.2%)	1 (5.5%)	3 (16.66%)	6 (33.3%)
Total	53 (27.15%)	12 (22.64)	9 (16.98)	13 (24.52%)
Absent	142 (72.8%)	-	3 (2.1%)	5 (3.5%)

In both groups (195 cases), notch was seen in 53 cases (27.17%). Among them 25 cases had HTD (47.16%), 21 cases (39.62%) had FGR, 12 cases had FGR with HTD (22.64%).

6. NOTCH AND HTD:-

Table X-A

Group - I

NOTCH	HTD		Total
	Absent	Present	
Absent	73	2	75
Bilateral	7	14	11
Unilateral	9	2	11
Total	89	8	97

P= 0.000

Conclusion: P- value of 0.000 indicates significant relationship between notch and hypertensive disorder.

Table - X-B

Group - II

NOTCH	HTD		Total
	Absent	Present	
Absent	64	3	67
Bilateral	10	14	24
Unilateral	2	5	7
Total	76	22	98

P= 0.000

Conclusion: P- value of 0.000 indicates significant relationship between notch and hypertensive disorder.

7. NOTCH AND FGR:-

Table XI-A

Group - I

NOTCH	FGR		Total
	Present	Absent	
Absent	1	74	75
Bilateral	3	8	11
Unilateral	2	9	11
Total	6	91	97

P = 0.000

Conclusion: P value of 0.000 indicates significant relationship between notch and FGR.

Table - XI-B

Group II

NOTCH	FGR		Total
	Present	Absent	
Absent	2	65	67
Bilateral	14	10	24
Unilateral	2	5	7
Total	18	80	98

P = 0.000

Conclusion: P value of 0.000 indicates significant relationship between notch and FGR.

8. NOTCH AND HTD-FGR:-

Table - XII-A

Group I

NOTCH	HTD-FGR		Total
	Absent	Present	
Absent	75	0	75
Bilateral	9	2	11
Unilateral	11	0	11
Total	95	2	97

P=0.239

Conclusion: P value of 0.239 indicates insignificant relationship between notch and HTD-FGR.

Table - XII-B

Group II

NOTCH	HTD-FGR		Total
	Absent	Present	
Absent	67	0	67
Bilateral	15	9	24
Unilateral	6	10	16
Total	88	9	98

P = 0.000

Conclusion

P-value of 0.000 indicate significant relationship between bilateral notch and HTD- FGR.

9. PERSISTENCE OF NOTCH AND HTD:-

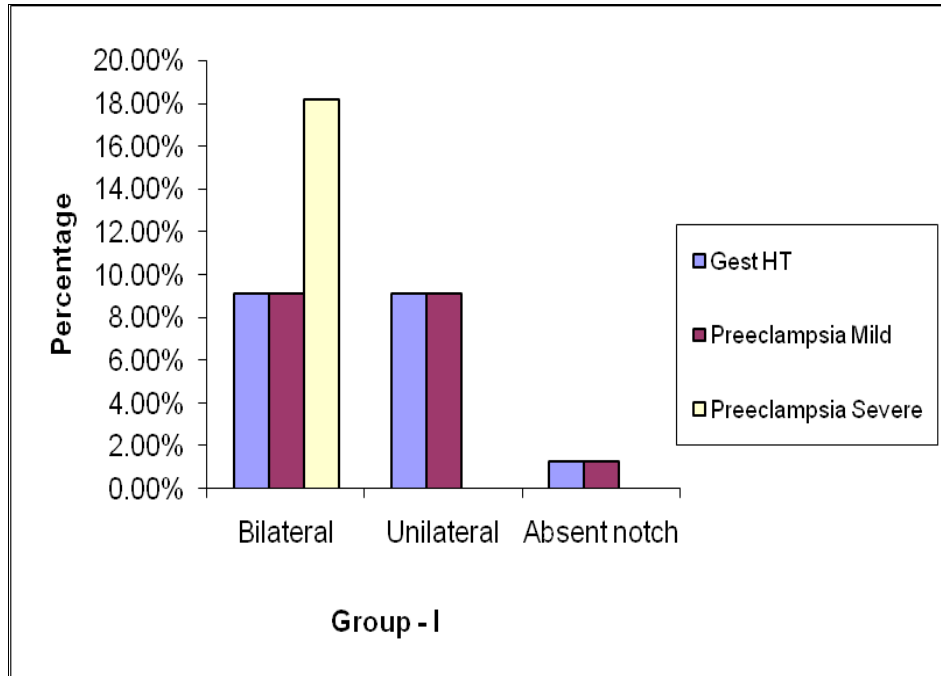
Table –XIII - A

Group - I

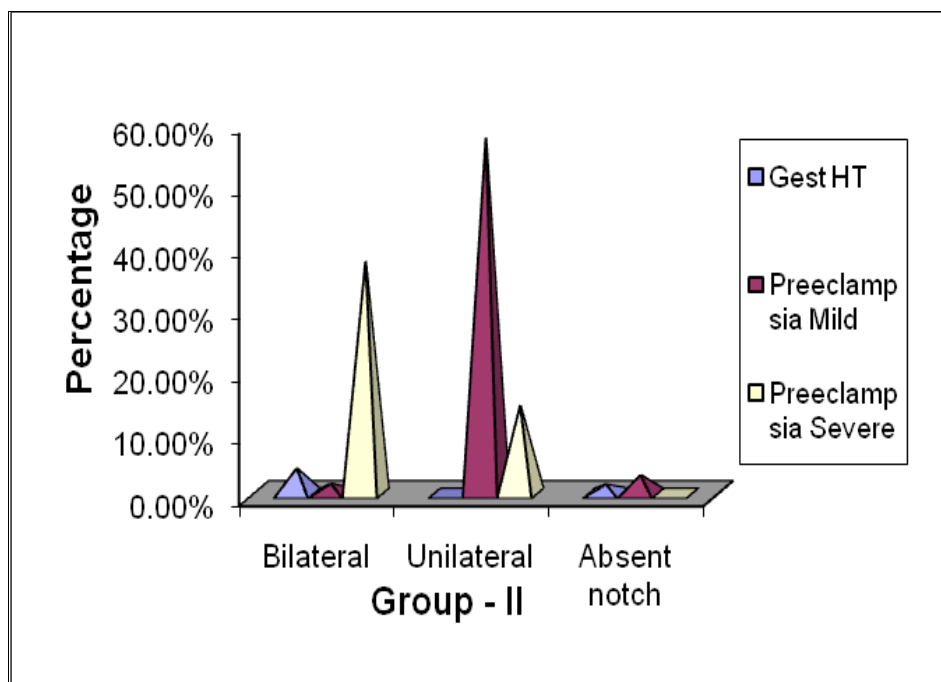
Notch	No of cases	Gest HT	Preeclampsia	
			Mild	Severe
Bilateral	11	1 (9.09%)	1 (9.09%)	2 (18.18%)
Unilateral	11	1 (9.09%)	1 (9.09%)	-
Total	22	2 (9.09%)	2 (9.09%)	2 (9.09%)
Absent notch	75	1 (1.3%)	1 (1.3%)	-

NOTCH AND HTD

Group - I



Group - II



In this study, in Group I in the cases with persistence of bilateral notch in 1 (5.7%) had gestational HT, 15 (14.28%) had mild preeclampsia, 2(31.42%) had severe preeclampsia.

In the cases with persistence of unilateral notch 1(5.5%) had gestational HT, 1 (27.77%) had mild preeclampsia,

Table – XIII - B

Group - II

Notch	No of cases	Gest HT	Preeclampsia	
			Mild	Severe
Bilateral	24	1 (4.1%)	4 (1.6%)	9 (37.5%)
Unilateral	7	-	4 (57.4%)	1 (14.28%)
Total	31	1 (3.2%)	8 (25.80%)	10 (32.25%)
Absent notch	67	1 (1.49%)	2 (2.98%)	-

In this study, in Group II in the cases of persistence of bilateral notch 1 (4.1%) had gestational HT, 4 (1.6%) had mild preeclampsia, 9(37.5%) had severe preeclampsia.

In the cases of unilateral notch 4 cases (57.4%) had mild preeclampsia, 1 case (14.28%) had severe preeclampsia.

10. MODE OF DELIVERY AND PERSISTENCE OF NOTCH:-

Table –XIV - A**Group - I**

Notch	No of cases	Mode of delivery	
		Vaginal	Caesarean
Bilateral	11	7 (63.63%)	4 (36.36%)
Unilateral	11	9 (81.81%)	2 (18.18%)
Total	22	16 (72.72%)	6 (27.27%)
Absent notch	75	63 (84%)	12 (16%)

In Group I, in the presence of bilateral notch, 7 cases (63.63%) had vaginal delivery and 4 cases (36.36%) had caesarean delivery.

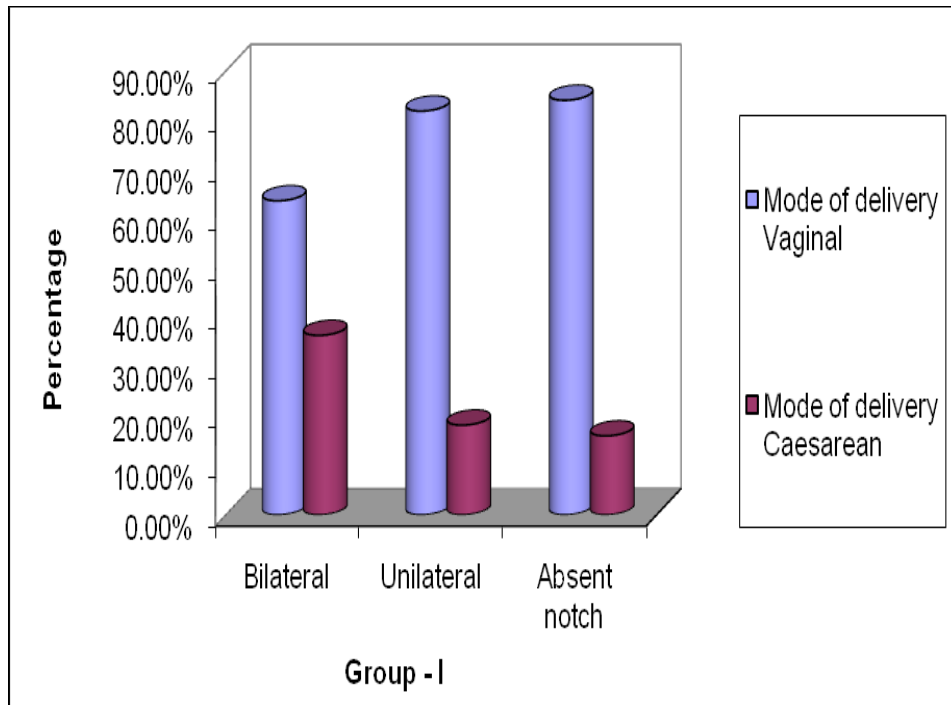
In the presence of unilateral notch, 9 cases (81.81%) had vaginal delivery and 2 cases (18.18%) had caesarean delivery.

Table –XIV – B**Group 1I**

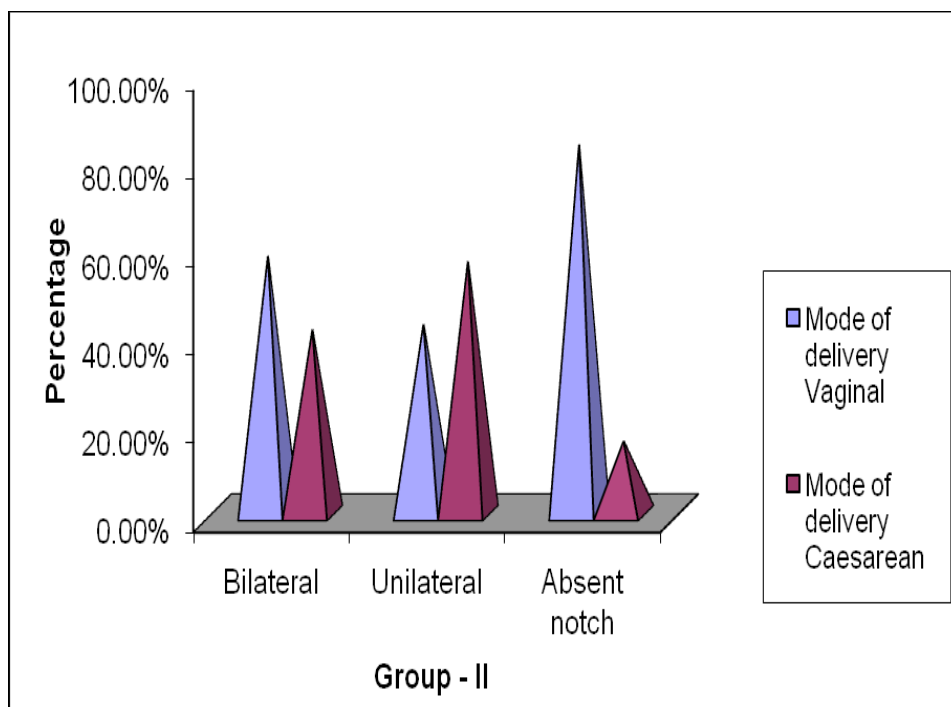
Notch	No of cases	Mode of delivery	
		Vaginal	Caesarean
Bilateral	24	14 (58.33%)	10 (41.66%)
Unilateral	7	3 (42.85%)	4 (57.14%)
Total	31	17 (54.83%)	14 (45.16%)
Absent notch	67	56 (83.58%)	11 (16.41%)

MODE OF DELIVERY AND PERSISTENCE OF NOTCH

Group - I



Group II



In Group II, in the presence of bilateral notch, 14 cases (58.33%) had vaginal delivery and 10 cases (41.66%) had caesarean delivery.

In the presence of unilateral notch, 3 cases (42.85%) had vaginal delivery and 4 cases (57.14%) had caesarean delivery.

11. PERSISTENCE OF NOTCH AND GESTATIONAL AGE AT DELIVERY:-

Table –XV – A

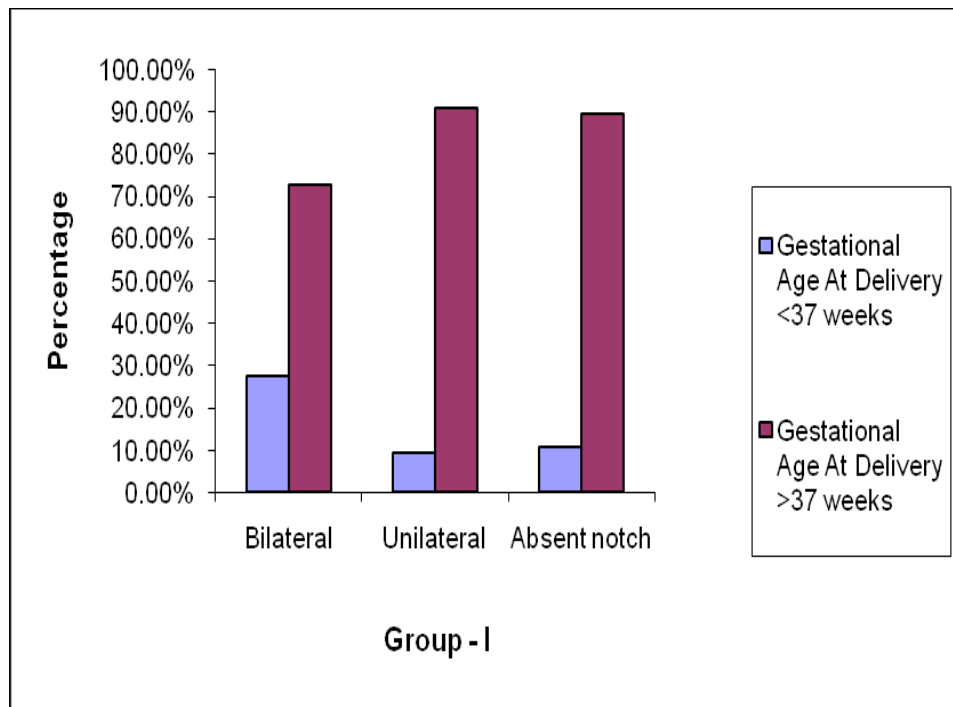
Group - I

Notch	No of cases	Gestational Age At Delivery	
		<37 weeks	>37 weeks
Bilateral	11	3 (27.27%)	8 (72.72%)
Unilateral	11	1 (9.09%)	10 (90.9%)
Total	22	4 (18.18%)	18 (81.81%)
Absent notch	75	8 (10.66%)	67 (89.33%)

In Group I, in the presence of bilateral notch, 3 cases (27.27%) had delivery at less than 37 weeks of gestation and in the presence of unilateral notch, 1 case (9.09%) had delivery at less than 37 weeks of gestation.

PERSISTENCE OF NOTCH AND GESTATIONAL AGE AT DELIVERY

Group - I



Group II

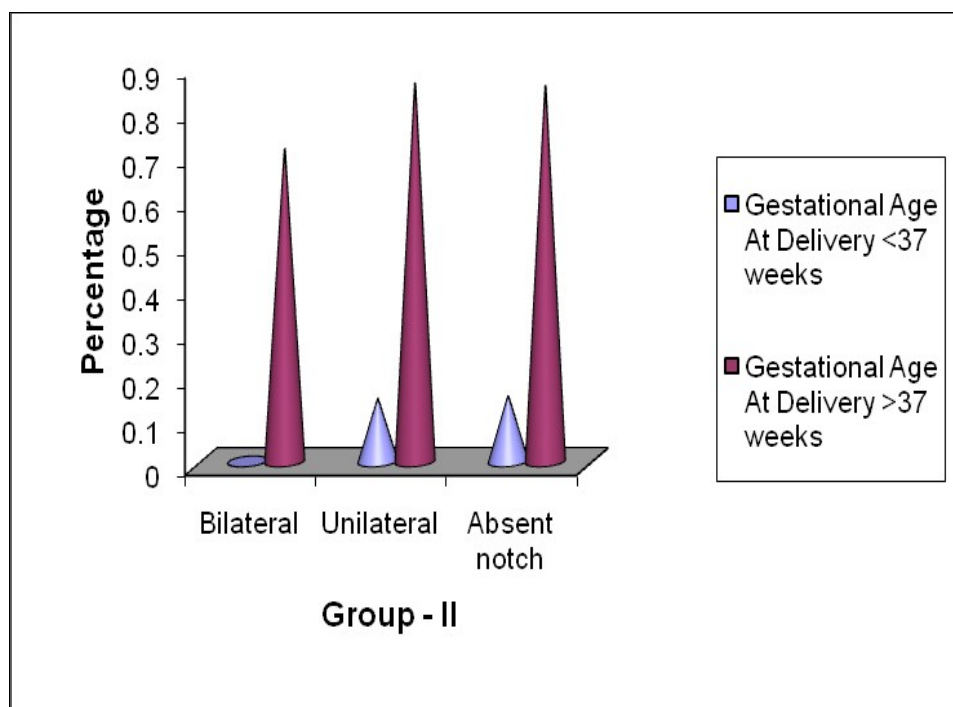


Table –XV – B**Group - II**

Notch	No of cases	Gestational Age At Delivery	
		<37 weeks	>37 weeks
Bilateral	24	7 (29.16%)	17 (70.83%)
Unilateral	7	1 (14.28%)	6 (85.71%)
Total	31	8 (25.8%)	23 (74.19%)
Absent notch	67	10 (14.92%)	57 (85.07%)

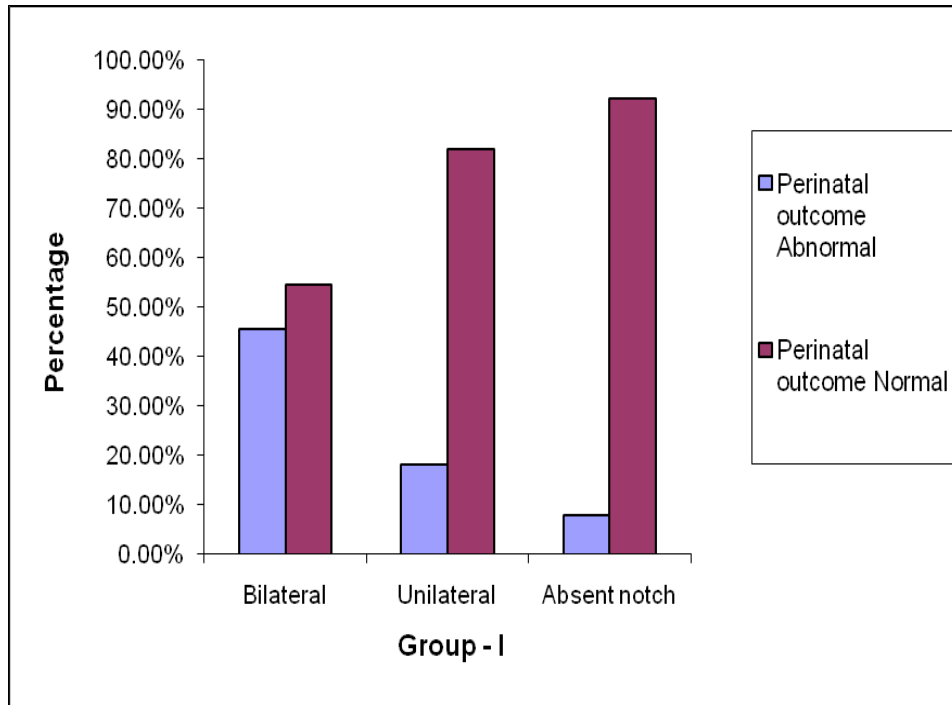
In Group II, in the presence of bilateral notch, 7 cases (29.16.%) had delivery at less than 37 weeks of gestation and in the presence of unilateral notch 1 case (14.28%) had delivery at less than 37 weeks of gestation.

13. PERSISTENCE OF NOTCH AND PERINATAL OUTCOME:-**Table –XVI - A****Group - I**

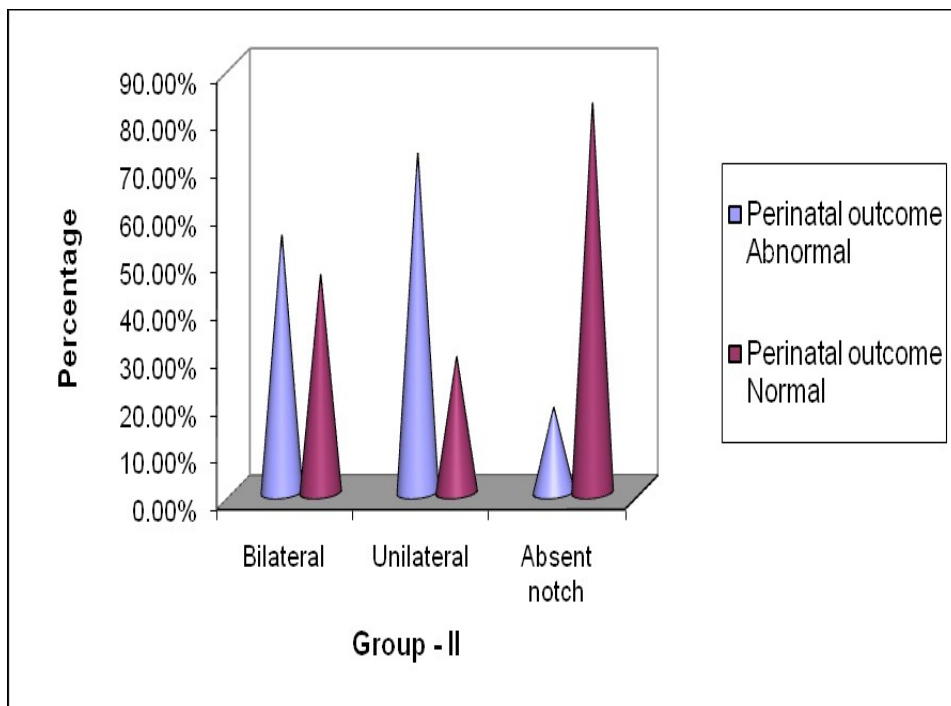
Notch	Number of cases	Perinatal outcome	
		Abnormal	Normal
Bilateral	11	5 (45.45%)	6 (54.54%)
Unilateral	11	2 (18.18%)	9 (81.81%)
Total	22	7 (31.81)	15 (68.18%)
Absent notch	75	6 (8%)	69 (92%)

PERSISTENCE OF NOTCH AND PERINATAL OUTCOME.

Group - I



Group - II



In Group I, in the presence of bilateral notch, 5 cases (45.45%) had abnormal perinatal outcome and in the presence of unilateral notch 2 cases (18.18%) had abnormal perinatal outcome.

Abnormal perinatal outcome was noted as apgar <7/10, Meconium aspiration syndrome, respiratory distress, small for gestational age, preterm delivery and its complications, NICU admission.

Table –XVI - B

Group II

Notch	Number of cases	Perinatal outcome	
		Abnormal	Normal
Bilateral	24	13 (54.16%)	11 (45.83%)
Unilateral	7	5 (71.42%)	2 (28.57%)
Total	31	18 (58.06%)	13 (41.93%)
Absent notch	67	12 (17.9%)	55 (82.03%)

In Group II, in the presence of bilateral notch, 13 cases (54.16%) had abnormal perinatal outcome and in the presence of unilateral notch 5 cases (71.42%) had abnormal perinatal outcome.

14. NOTCH AND MODE OF DELIVERY:-

Table –XVII - A**Group I**

NOTCH	Mode of delivery		Total
	Caesarean	Vaginal	
Absent	10	65	75
Bilateral	3	8	11
Unilateral	1	10	11
Total	14	83	97

P = 0.006

Conclusion

P-value of 0.006 indicate significant relationship between notch and mode of delivery. Bilateral and unilateral notch are associated with increased incidence of caesarean section.

Table –XVII - B**Group II**

NOTCH	Mode of Delivery		Total
	Caesarean	Vaginal	
Absent	13	54	67
Bilateral	11	13	24
Unilateral	5	2	7
Total	29	69	98

P = 0.000

Conclusion

P-value of 0.000 indicate significant relationship between notch and mode of delivery. Bilateral and unilateral notch are associated with increased incidence of caesarean section.

15. NOTCH AND GESTATIONAL AGE AT DELIVERY:-

Table –XVIII - A

Group I

NOTCH	Gestational age		Total
	>37wks	<37 wks	
Absent	70	5	75
Bilateral	9	2	11
Unilateral	11	0	11
Total	90	7	97

P = 0.239

Conclusion

P-value of 0.239 indicate an insignificant relationship between notch and gestational age at delivery.

Table –XVIII - B

Group II

NOTCH	Gestational age		Total
	>37 wks	<37 wks	
Absent	64	3	67
Bilateral	18	6	24
Unilateral	6	1	7
Total	88	10	98

P = 0.016

Conclusion

P-value of 0.016 (only at 5%) indicates a significant relationship between notch and gestational age at delivery. Gestational age <37 wks is associated with bilateral notch.

17. NOTCH AND PERINATAL OUTCOME:-

Table –XIX - A

Group I

NOTCH	Perinatal outcome		Total
	Abnormal	Normal	
Absent	10	57	67
Bilateral	12	12	24
Unilateral	3	4	7
Total	25	73	98

P=0.012

Conclusion

P-value of 0.012 (only at 5%) indicate a significant relationship between notch and perinatal outcome. Abnormal Perinatal outcome is associated with Unilateral and Bilateral Notch.

Table –XIX - B

Group II

NOTCH	Perinatal out come		Total
	Abnormal	Normal	
Absent	8	67	75
Bilateral	6	5	11
Unilateral	4	7	11
Total	18	79	97

P=0.037

Conclusion

P-value of 0.037 indicate significant(at 5%) relationship notch and perinatal outcome. Notch is associated with perinatal abnormality.

Evaluation of Diagnostic Test

The term validity refers to what extent the test accurately measures what it purposes to measure. Validity has 2 components viz sensitivity and specificity

Doppler study

		Notch	
		+	-
Out come	+	TP ^a	FN ^c
	-	FP ^b	TN ^d

Following measures are used to evaluate a screening tests,

$$1) \text{ Sensitivity} = \frac{a}{a+c} \times 100$$

$$2) \text{ Specificity} = \frac{d}{b+d} \times 100$$

$$3) \text{ Positive predictive value of the test} = \frac{a}{a+b} \times 100$$

$$4) \text{ Negative predictive value of the test} = \frac{d}{c+d} \times 100$$

$$5) \text{ Likelihood ratio (+)test} = \frac{\text{Sensitivity}}{1-\text{Specificity}}$$

$$6) \text{ Likelihood ratio (-) test} = \frac{1-\text{Sensitivity}}{\text{Specificity}}$$

$$7) \text{ Percentage of false positive} = \frac{b}{b+d} \times 100$$

$$8) \text{ Percentage of false negative} = \frac{c}{a+c} \times 100$$

Group I

1. Any notch for HTD

		Notch		
		+	-	
HTD	+	6	2	8
	-	16	73	89
		22	75	97

$$1 \quad \text{Sensitivity} = \frac{6}{8} \times 100 = 75\%$$

$$2 \quad \text{Specificity} = \frac{73}{89} \times 100 = 82.02\%$$

$$3 \quad \text{PPV} = \frac{6}{22} \times 100 = 27.27\%$$

$$4 \quad \text{NPV} = \frac{73}{75} \times 100 = 97.33\%$$

$$5 \quad \text{Likelihood ratio for positive test} = \frac{0.75}{1 - 0.82} = 4.16$$

$$6 \quad \text{Likelihood ratio for negative test} = \frac{1 - 0.75}{0.82} = 0.3$$

2. Bilateral notch for HTD

Bilateral Notch

		+	-	
	+	4	2	6
HTD	-	7	84	91
		11	86	97

$$1 \quad \text{Sensitivity} = \frac{4}{6} \times 100 = 66\%$$

$$2 \quad \text{Specificity} = \frac{84}{91} \times 100 = 92\%$$

$$3 \quad \text{PPV} = \frac{4}{11} \times 100 = 36\%$$

$$4 \quad \text{NPV} = \frac{84}{86} \times \frac{10}{0} = 97.67\%$$

$$5 \quad \text{Likelihood ratio for positive test} = \frac{0.66}{1-0.92} = 8.25$$

$$6 \quad \text{Likelihood ratio for negative test} = \frac{1-0.66}{0.92} = 0.36$$

3.Any notch for FGR

		Notch		
		+	-	
FGR	+	5	1	6
	-	17	74	91
		22	75	97

$$1 \quad \text{Sensitivity} = \frac{5}{6} \times 100 = 83.33\%$$

$$2 \quad \text{Specificity} = \frac{74}{91} \times 100 = 81.31\%$$

$$3 \quad \text{PPV} = \frac{5}{22} \times 100 = 22.72\%$$

$$4 \quad \text{NPV} = \frac{74}{75} \times 100 = 98.66\%$$

$$5 \quad \begin{array}{l} \text{Likelihood} \\ \text{ratio for} \\ \text{positive} \\ \text{test} \end{array} = \frac{0.83}{1-0.81} = 4.36$$

$$6 \quad \begin{array}{l} \text{Likelihood ratio} \\ \text{for negative test} \end{array} = \frac{1-0.83}{0.81} = 0.2$$

4. Bilateral notch for FGR

Bilateral Notch

		+	-	
FGR	+	3	1	4
	-	8	85	93
		11	86	97

$$1 \quad \text{Sensitivity} = \frac{3}{4} \times 100 = 75\%$$

$$2 \quad \text{Specificity} = \frac{85}{93} \times 100 = 91.39\%$$

$$3 \quad \text{PPV} = \frac{3}{11} \times 100 = 27.27\%$$

$$4 \quad \text{NPV} = \frac{85}{86} \times 100 = 98.83\%$$

$$5 \quad \begin{array}{l} \text{Likelihood} \\ \text{ratio for} \\ \text{positive} \\ \text{test} \end{array} = \frac{0.75}{1-0.91} = 8.3$$

$$6 \quad \begin{array}{l} \text{Likelihood ratio} \\ \text{for negative test} \end{array} = \frac{1-0.75}{0.91} = 0.27$$

Group II

1. Any notch for HTD

Any Notch

		+	-	
HTD	+	19	3	22
	-	12	64	76
		31	67	98

$$1 \quad \text{Sensitivity} = \frac{19}{22} = 86.36\%$$

$$2 \quad \text{Sensitivity} = \frac{86}{90} = 84.21\%$$

$$3 \quad \text{PPV} = \frac{7}{11} = 61.29\%$$

$$4 \quad \text{NPV} = \frac{86}{86} = 95.52\%$$

$$5 \quad \begin{array}{l} \text{Likelihood} \\ \text{ratio for} \\ \text{positive} \\ \text{test} \end{array} = \frac{86.36}{100-84.21} = 5.46$$

$$6 \quad \begin{array}{l} \text{Likelihood} \\ \text{ratio for} \\ \text{negative} \\ \text{test} \end{array} = \frac{100-86.36}{84.21} = 0.16$$

2. Bilateral notch for HTD

Bilateral Notch

		+	-	
HTD	+	14	3	17
	-	10	71	81
		11	86	98

$$1 \quad \text{Sensitivity} = \frac{14}{17} = 82.35\%$$

$$2 \quad \text{Sensitivity} = \frac{71}{81} = 87.65\%$$

$$3 \quad \text{PPV} = \frac{14}{24} = 58.33\%$$

$$4 \quad \text{NPV} = \frac{71}{74} = 95.94\%$$

$$5 \quad \begin{array}{l} \text{Likelihood} \\ \text{ratio for} \\ \text{positive} \\ \text{test} \end{array} = \frac{82.35}{100-87.65} = 6.66$$

$$6 \quad \begin{array}{l} \text{Likelihood} \\ \text{ratio for} \\ \text{negative test} \end{array} = \frac{100-82.35}{87.65} = \frac{17.65}{87.65} = 0.2$$

3. Any notch for FGR

		Notch		
		+	-	
FGR	+	16	2	18
	-	15	65	80
		31	67	98

$$1 \quad \text{Sensitivity} = \frac{16}{18} = 88.88\%$$

$$2 \quad \text{Sensitivity} = \frac{65}{80} = 81.25\%$$

$$3 \quad \text{PPV} = \frac{16}{31} = 51.61\%$$

$$4 \quad \text{NPV} = \frac{65}{67} = 97.01\%$$

$$5 \quad \begin{array}{l} \text{Likelihood} \\ \text{ratio for} \\ \text{positive} \\ \text{test} \end{array} = \frac{88.88}{100 - 81.25} = 4.74$$

$$6 \quad \begin{array}{l} \text{Likelihood} \\ \text{ratio for} \\ \text{negative} \\ \text{test} \end{array} = \frac{100 - 88.88}{81.25} = \frac{11.12}{81.25} = 0.13$$

4. Bilateral notch for FGR

Bilateral Notch

		+	-	
FGR	+	14	2	16
	-	10	72	82
		24	74	98

$$1 \quad \text{Sensitivity} = \frac{14}{16} = 87.5\%$$

$$2 \quad \text{Sensitivity} = \frac{72}{82} = 87.80\%$$

$$3 \quad \text{PPV} = \frac{14}{24} = 58.3\%$$

$$4 \quad \text{NPV} = \frac{72}{74} = 97.29\%$$

$$5 \quad \begin{array}{l} \text{Likelihood} \\ \text{ratio for} \\ \text{positive} \\ \text{test} \end{array} = \frac{87.5}{100-87.8} = 7.15$$

$$6 \quad \begin{array}{l} \text{Likelihood} \\ \text{ratio for} \\ \text{negative} \\ \text{test} \end{array} = \frac{100-87.5}{87.8} = 0.14$$

PREDICTION OF HTD /FGR BY UTERINE ARTERY DOPPLER SCREENING

For Group - I

Diagnosti c Test	Sensi	Speci	PPV	NPV	LR for + test (95% CI)	LR for (-) Test (95% CI)	FP	FN
For HTD								
Any notch	75%	82.0%	27.27 %	97.33%	4.16	0.3	6.7%	25%
Bilateral notch	66.%`	92%	36%	97.67%	8.25	0	7.6%	33.3 %
For FGR								
Any notch	83.33 %	81.3%	22.72 %	98.66%	4.36	0.2	18.68 %	16.6 %
Bilateral notch	75%	91.39 %	27.27 %	98.83%	8.3	0.27	8.6%	25%

For Group – II

Diagnostic Test	Sensi	Speci	PPV	NPV	LR for + test (95% CI)	LR for (-) Test (95% CI)	FP	FN
For HTD								
Any notch	86.36%	84.21%	61.29%	95.52%	5.46	0.16	15.78%	13.6%
Bilateral notch	86.35%	87.65%	58.33%	95.94%	6.66	0.2	12.34%	21.4%
For FGR								
Any notch	88.88%	81.25%	51.61%	97.0%	4.74	0.13	18.75%	11.1%
Bilateral notch	87.5%	87.80%	58.3%	97.29%	7.17	0.14	12.1%	12.5%

DISCUSSION

DISCUSSION

Doppler velocimetry is a noninvasive technic which uses high frequency sound for the investigation of blood flow. The feasibility of its fetal application was first reported by Fitzgerald and Drumm³⁷. It made non invasive investigation of utero placental circulation possible. Diastolic notch is defined as the slower velocity just after systolic flow but before maximum diastolic flow.

In this study, there was statistically significant association between the persistence of both unilateral, bilateral notching and development of hypertensive disorders of pregnancy, fetal growth restriction when compared to notch absent groups.

In Group I, 36.36% had HTD of pregnancy and 27.27% had FGR in the presence of bilateral notch, 18.18% had HTD and 18.18% had FGR in the presence of unilateral notch. In Group II, 58.33% had HTD and 58.33% had FGR in the presence of bilateral notch and 71.4% had HTD and 28.57% had FGR in the presence of unilateral notch.

Persistence of bilateral notching was associated significantly with severe forms of hypertensive disorders of pregnancy (31.42%) when compared to unilateral notching (5.5%).

A prospective trial of Zimmermann et al⁴³ evaluated the utility of uterine artery doppler between 21-24 wks in the prediction of preeclampsia and FGR. He selected 172 low risk pregnancies and 175 women at risk for hypertensive disorders of pregnancy /FGR. Presence of persistent notch accounted for 3-4 fold increased risk in developing preeclampsia /FGR. In this group, preeclampsia /FGR was found in 58.3% compared to 8.3% if doppler results were normal. Doppler was less informative in low risk population. Here preeclampsia /FGR were 6.1-6.4% in this low risk group and 5.2% in notch absent group.

Deutinger et al²⁸ believed that early diastolic notch persistence was thought to represent the persistence of inherent total impedance of the uteroplacental circulation.

Rofinas et al³² found that the persistence of uterine artery diastolic notch indicates severe hypertensive disorder and associated with increased rate of FGR, caesarean delivery, fetal distress and preterm delivery.

In 1983, Campbell et al³⁹ was the first to demonstrate a correlation between pregnancies complicated by hypertensive disorder /FGR, increased caesarean rate, fetal distress, low APGAR scores and persistence of uterine artery notch. Furthermore, proteinuria and severe hypertension correlated significantly with persistent notch.

Flesicher et al¹¹ in 1986 demonstrated the presence of an early diastolic notch in the uterine artery after 26 weeks gestation correlated significantly with the clinical diagnosis of preclampsia, abnormal perinatal outcome, increased caesarean rate.

Trudinger¹² in 1990 did doppler uterine artery in a highly selected population for prediction of severe PIH.

Thaler²² et al, demonstrated the persistence of an early diastolic notch after 26 weeks of gestation in 25-40% of preeclampsia. Presence of notch is significantly a better predictor of poor pregnancy than the S/D ratio (or) resistive index.

Pai³⁶ found persistent diastolic notch to be a better parameter than abnormal RI in predicting the hypertensive disorders of pregnancy / fetal growth restriction.

Bower³⁵ et al also predicted the hypertensive disorders of pregnancy / fetal growth restriction by persistence of uterine artery notch.

In this study, presence of notch was significantly associated with increased rate of caesarean section when compared to notch absent cases. In the presence of bilateral notch, 36.36% cases and in the presence of unilateral notch 18.18% cases, in notch absent cases 16% had caesarean

section in group I . In the presence of bilateral notch 41.66%, in the presence of unilateral notch 57.14% and 16.41% in the notch absent cases had caesarean section. Incidence of caesarean section was more in high risk cases.

In this study, rate of preterm delivery was more in the presence of bilateral notch (28.57%) when compared to unilateral notch (11.11%) and absence of notch (12.67%).

Aristidou et al²⁷ noted that the uterine artery notch was a good predictor of poor perinatal outcome, increased rate of FGR, caesarean delivery for fetal distress, preterm delivery.

In 2001, Christopher Lees³⁴ carried out a colour doppler assessment of uterine artery in 5121 women attending routine antenatal clinic and concluded that persistent uterine artery notch associated with adverse perinatal outcome.

Albaiges et al 2006 ³⁸ had sensitivity of 70% for prediction of preeclampsia and FGR. But the positive predictive value was <10%. In this study, positive predictive value is low in group I when compared to group II which limits its usage in the general population.

Validity of tests in Group I&II for any notch and bilateral notch for hypertensive disorder/ fetal growth restriction when compared to other studies were,

For prediction of PIH

Author	Sensitivity	Specificity	Positive predictive value	Negative predictive value
Bower et al ³⁵	78	96	28	99.5
Pai ³⁶	45.45	92	38	93.87
Agarwal	84	71.4	72	-
May Backos et al	38	85	27	90
Campbell et al	68%	69%	-	-
Papageorgiou et al	41%	-	-	-

In this study,

Group I

Notch	Sensitivity	Specificity	Positive predictive value	Negative predictive value	LR for + test	LR for – test	FP	FN
Any notch	75%	82.0%	27.27%	97.33%	4.16	0.3	6.7%	25%
Bilateral	66%	92%	36%	97.67%	8.25	0.36	7.61 %	33.3%

Group II

Notch	Sensitivity	Specificity	Positive predictive	Negative predictive	LR for	LR for	FP	FN
-------	-------------	-------------	---------------------	---------------------	--------	--------	----	----

			value	value	+ test	– test		
Any notch	86.36%	84.21%	61.29%	95.52%	5.46	0.16	15.78 %	13.63 %
Bilateral	82.34%	87.65%	58.33%	95.94%	6.66	0.2	12.34 %	21.42 %

For prediction of FGR

Author	Sensitivity	Specificity	Positive predictive value	Negative predictive value
May Backos et al ⁴⁵	41%	85%	30%	90%
Papageorghiou et al ⁴⁶	24%	-	-	-

Group I

Notch	Sensi	Speci	Positive predictive value	Negative predictive value	LR for + test	LR for – test	FP	FN
Any notch	83.33%	81.31%	22.7%	98.66%	4.36	0.2	18.68 %	16.66%
Bilateral	75%	91.39%	27.27%	98.83%	8.3	0.2 7	8.6%	25%

Group II

Notch	Sensi	Speci	Positive predictive value	Negative predictive value	LR for + test	LR for – test	FP	FN
-------	-------	-------	---------------------------	---------------------------	---------------	---------------	----	----

Any notch	88.8%	81.25%	51.61%	97.01%	4.74	0.13	18.75%	11.11%
Bilateral	87.5%	87.80%	58.3%	97.29%	7.17	0.14	12.1%	12.5%

The most useful part of the test is the negative predictive value. A negative test at 24 wks in a high risk population indicates a 97-99% probability that HTD/ FGR will not be present³⁶. So in the absence of notch, reassurance can be given to the high risk cases.

Valensise et al 1993³⁸ has better sensitivity of 88% for the prediction of preeclampsia. Conde- Agudelo et al 1993⁴¹ found that the sensitivity of the test was 72-92% in prediction of hypertensive disorders of pregnancy. On evaluating the likelihood ratio for positive and negative tests, presence of any notch is mild predictor of these disorders in group I and was moderate predictor of hypertensive disorders / fetal growth restriction in group II.

It can be evaluated along with routine scan in all women if possible. But in high risk women it should be specifically evaluated for better antenatal care so that necessary timely intervention can be done.

Several factors are likely to influence the performance of screening tests and these include anatomical site of measurement of uterine artery doppler. Test to be done at standard reference point⁴² for better prediction. Rigid definition for outcomes to be used for the proper study.

SUMMARY

SUMMARY

IN GROUP - I

- ❖ 68% of cases belonged to 21- 30yrs of age
- ❖ 68.04% of cases were primigravida and 31.96% were multigravida.
- ❖ Bilateral notch was present in 11.34% of cases (primi- 81.81%, multi- 18.18%)
- ❖ Unilateral notch was present in 11.34% of cases (primi – 72.72%, multi – 27.27%).
- ❖ 36.36% of cases had hypertensive disorders of pregnancy, 27.27% of cases had fetal growth restriction, 18.18% of cases had hypertensive disorder with fetal growth restriction, 36.36% had caesarean delivery, 27.27% of cases had preterm delivery, 45.45% had abnormal perinatal outcome in the presence of bilateral notch.
- ❖ In cases with unilateral notch, 18.18% of cases had hypertensive disorders of pregnancy, 18.18% of cases had fetal growth restriction, 18.18% had caesarean delivery, 9.09% had preterm delivery, 18.18% had abnormal perinatal outcome.
- ❖ In the absence of notch, 2.6% of cases had hypertensive disorders, 1.3% cases had fetal growth restriction, 16% had caesarean section, 10.66% had preterm delivery, 8% had abnormal perinatal outcome.

- ❖ There is significant association between notch and HTD, FGR, abnormal perinatal outcome, mode of delivery and no significant association between notch and HTD with FGR, gestational age at delivery. In the presence of notch, there is increased incidence of HTD, FGR, caesarean delivery, preterm delivery, abnormal perinatal outcome.

Group I

Diagnostic Test	Sensi	Speci	PPV	NPV	LR for + test (95% CI)	LR for (-) Test (95% CI)	FP	FN
For HTD								
Any notch	75%	82.0%	27.27%	97.33%	4.16	0.3	6.7%	25%
Bilateral notch	66.7%	92%	36%	97.67%	8.25	0	7.6%	33.3%
For FGR								
Any notch	83.33%	81.3%	22.72%	98.66%	4.36	0.2	18.68%	16.6%
Bilateral notch	75%	91.39%	27.27%	98.83%	8.3	0.27	8.6%	25%

IN GROUP II

- ❖ 77.5% of cases belonged to the age group of 21 to 30 years of age.
- ❖ 100% were multigravida.
- ❖ Bilateral notch was present in 24.49% of cases
- ❖ Unilateral notch was present in 7.14% of cases.
- ❖ In the presence of bilateral notch, 58.33% had hypertensive disorders, 58.33% had fetal growth restriction, 37.5% had hypertensive

disorders with fetal growth restriction, 41.66% had caesarean delivery, 16% had preterm delivery, 54.16% had abnormal perinatal outcome.

- ❖ In the presence of unilateral notch, 71.4% had hypertensive disorders, 28.57% had fetal growth restriction, 14.28% had hypertensive disorders with fetal growth restriction, 57.14% had caesarean delivery, 14.28% had preterm delivery, 71.42% had abnormal perinatal outcome.
- ❖ There is significant association between notch and HTD, FGR, abnormal perinatal outcome, mode of delivery, HTD with FGR, gestational age at delivery. In the presence of notch, there is increased incidence of HTD, FGR, caesarean delivery, preterm delivery abnormal perinatal outcome

Group II

Diagnostic Test	Sensi	Speci	PPV	NPV	LR for + test (95% CI)	LR for (-) Test (95% CI)	FP	FN
For HTD								
Any notch	86.36%	84.21%	61.29%	95.52%	5.46	0.16	15.78%	13.6%
Bilateral notch	86.35%	87.65%	58.33%	95.94%	6.66	0.2	12.34%	21.4%
For FGR								
Any notch	88.88%	81.25%	51.61%	97.0%	4.74	0.13	18.75%	11.1%
Bilateral notch	87.5%	87.80%	58.3%	97.29%	7.17	0.14	12.1%	12.5%

CONCLUSION

CONCLUSION

- 1) In group I, persistent uterine artery notch is a mild predictor of development of hypertensive disorders of pregnancy and fetal growth restriction.
- 2) In group II, persistent uterine artery notch is a moderate predictor of development of these disorders. Presence of bilateral notch is significantly associated with severe form of hypertensive disorders.
- 3) In high risk pregnancies, an abnormal uterine artery doppler is an indication for a closer antenatal follow up and normal uterine artery doppler is reassuring and allows less frequent fetal surveillance when compared to positive test.
- 4) To improve the predictive value of tests, it can be combined with clinical high risk factors, also with estimation of serum inhibin A, β HCG concentration.
- 5) In developing countries like India, cost effectiveness of the tests should also be taken into consideration.
- 6) Hence in high risk women, persistence of uterine artery notch especially bilateral notch should be specifically evaluated so that necessary timely intervention can be made. Persistent uterine artery notch in high risk women is a good predictor of hypertensive disorders of pregnancy and FGR.

BIBLIOGRAPHY

BIBLIOGRAPHY

1. James 3rd edition, High risk pregnancy, Fetal growth disorder P.No. 240-264
2. Schulman H, Ducey J, Farmakides G et al uterine art. doppler velocimetry Am J obst Gynaec157:1539, 1987.
3. Edwin R. Gudman Doppler Velocimetry of the uteroplacental circulation – Doppler USG in obst /Gynae chap 12:193-224.
4. Fleischer A, Schulman H. Farmakides G et al; uterine art Doppler velocimetry in pregnant women with HT. Am J. Obstetric Gynaecology 1986;154:806.
5. Williams Obstetrics 22nd Edition USG and Doppler P:No.389-406.
6. Christopher R. Harman and Ahmet :Arterial and venous doppler in IUGR. Clinical obs and Gyn. Vol 46 No.4: 931-936.
7. Muralidhar v Pai, uterine art. doppler velocimetry in women with normal pregnancy, PIH and IUGR. Obs and Gyn. Today vol VI No11: Nov.2001.
8. Fleischer A, Schulman H, Farmakides G et al., Uterine artery velocity waveforms and IUGR, Am J obs Gyn 1985; 151:502.
9. Walfrido W. Sumpaico, Management of preg induced HT Obs and Gyn for post graduates volume 1 : 56-57.

10. George Farmakides, Doppler velocimetry and HT: Doppler USG in obs and gyn chap 15;286-293.
11. Wladim Sroff JW, Tonge HM, Stewart P.A. Doppler USG assessment of uterine art flow in fetus. Br J obs Gynaecol 1986; 93:470-475.
12. Trudinger B, Cook CM: umbilical and uterine art flow velocity wave forms in pregnancy associated with major congenital abnormality Br J obs gyn1985; 92:666.
13. Campbell S. Thomas A. ultrasonic measurement of the fetal head to Abdominal circumference ratio in the assessment of growth restriction. Br J obs and Gyn 1977; 84: 165
14. Dev maulik. Doppler for clinical management what is its place? Obs and gyn clinics of NA vol 18, No. 4 Dec 1991.
15. Neilson: Doppler USG in Highrisk Pregnancies systemic review with meta analysis: Am J obs Gyn. 1995; 172: 1379- 1357.
16. Arduini D, Rizzo G, Romaninic, et al., fetal blood flow velocity wave forms as predictor of growth retardation obs gyn 1987; 70:7.
17. Arduini D, Rizzo G, Romaninic, et al., uteroplacental blood flow velocity wave forms as predictor of PIH. Eur J obs and Gyn Reprod 1987; 26:325, 1987.

18. R Holmes the aetiology of SGA – Progress in obs and Gyn by John stud Vol 13.
19. Dev maulik ; physical principles of Doppler USG – Doppler USG in obs & gyn chapter 2 ; 9-20.
20. Fernando Arias .The use of Doppler wave form analysis in the evaluation of the highrisk fetus. Obs and Gyn clinics of North America volume 15, No2, June 1988.
21. Hanretty KP, Whittle MJ; Doppler uteroplacental wave forms in PIH Lancet L: 850, 1988.
22. Israel Thaler and Amit: Doppler velocimetry of the uteroplacental circulation during early preg – Doppler USG in obs and Gyn chapter 13, 229-252.
23. Khong TY, Dewolf F, Robertson WB et al., Inadequate maternal vascular response to placentation in pregnancies complicated by preeclampsia and by SGA infants. Br J obs Gyn 1986; 1049.
24. Muralidhar V Pai, Rebecca Lama: The effect of placental laterality on uterine art Doppler velocimetry in normal pregnancy, PIH and IUGR Obs and Gyn of India. vol 52, No 1. Jan / feb. 2002 pg 52-55.
25. Nidhikhosla, Usha manaktala, Sukbir singh. Role of colour doppler in IUGR fetus obs and Gyn. Today volume VI No. 2 Feb 2001.

26. Muralidhar V Pai, Uterine art. doppler velocimetry in normal pregnancy, PIH, IUGR. Obs and Gyn today Vol VI. No 11 Nov. 2001.
27. Siegfried Rotmensch, Joshua A, Copel John C Hobins; Introduction to doppler velocimetry in obs and Gyn clinics of North America – Vol 18, No 4 , Dec 1991.
28. Steel SA, Pearce J M., MC Parkland P : early Doppler USG screening in prediction of HT disorders of preg. Lancet 1990; 335: 1548.
29. Brosens I. The physiological response of the vessels of the placental bed to normal pregnancy J. pathol Bacteriol 1967; 93 ; 569-79.
30. Campbell S, et.al, New Doppler technique for assessing utero placental Blood flow Lancet 1983 : I ; 676-7.
31. Campbell. S. Qualitative assessment of uteroplacental blood flow: Early screening test for high risk pregnancies obs and Gyn 1986: 68: 649-53.
32. Haraldur M- Gudnason – Gudmundsson –Preeclampsia– Abnormal Uterine art. Doppler related to recurrence of sym during next preg. 10/1515/Jpm/ 2004, 138.
33. H. Li. H. Gudnason, Olofsson, M. Dubiel, S Gudmundsson – Univ. of .Lund, Dept. of Obs and Gyn, Malmo University, Hospital,

Sweden. Increased uterine artery Vascular impedance is related to adverse outcome of pregnancy. Jan 2005.

34. Neilson Malhotra, Jai deep Malhotra. Sakshi mittal : USG and Doppler evaluation obs and Gyn. Today vol VIII No. 4 April 2003.
35. Bower S, Bewley S, Campbell, S. Improved prediction of preeclampsia by 2 stage screening of the uterine arteries using the early diastolic notch and colour Doppler imaging. Obs Gyn 1993; 82 : 78-83.
36. Pai M.V improved prediction of PIH and IUGR by 2 stage screening of uterine artery Doppler velocimetry. Ind. J med USG 2001; 2:64-9.
37. Fitzgerald DE, Drumm E. Noninvasive measurement of human fetal circulation using USG; a new method BMJ 1977; 2: 1450 - 1.
38. Al baiges G, Missfelder – Lobos H, Leesc et al., one stage screening for pregnancy complications by color doppler assessment of gestation. Obs and Gyn 2000; 96: 559-64.
39. Campbell S. Bewley S, Cohen T. Investigation of the uteroplacental circulation by Doppler ultrasound perinatol 1987; 11: 362 -8.
40. Velansise H, Bezzecheri V, Rizzo G , et al., doppler velocimetry of the uterine artery as a screening test for gestational HT USG obs Gyn 1993 ; 3 : 18-22.

41. Conde – Agudelo A, Belizan JM, Lede R., et.al., what does an elevated mean arterial pressure in the 2nd half of pregnancy predict gestational HT / Preeclampsia? Am J obs Gyn 1993; 169: 509:514.
42. Jaeschke R, Guyatt GH, Sackett DL. Users guides to the medical literature III. How to use an article about a diagnostic test. B. what are the results and will they help me in caring for my patients? The Evidence – based medicine working group JAMA 1994; 271: 703-7.
43. Zimmermann P, Eirio V, Koskinen J et al, Doppler assessment of the uterine and uteroplacental circulation in the second trimester in pregnancies at high risk for preeclampsia or Intrauterine growth retardation; Comparison between different Doppler parameter, ultrasound obstetrics Gynaecology 1997; 9:330-338
44. Agarwal P, Agarwal Rajeev K. persistence uterine artery notch- A predictor of IUGR /PIH. J obs Gyn India vol: 56, No4: 2006. 301-303.
45. N.Venkat Raman, May Backos, uterine artery doppler in predicting pregnancy outcome in woman with APLS. Am College of obs and Gyn 2001. volume 98, No2, Aug 2001.
46. Papageorgiou At yu CKH, Bindra R et al. Multicenter screening to preeclampsia and FGR by transvaginal uterine artery Doppler at 23 wks of gestation USG Gyn 2001; 18:441-449.

47.GS Ghosh,S Gudmunsson uterine and umbilical artery Doppler are comparable in predicting postnatal outcome of growth restricted fetuses 2009. The Authors journal compilation RCOG 2009 BJOG An international Journal of obstetrics and Gynacology.

PROFORMA

PROFORMA

Name : Age : IP No: Unit:

Address: Parity: Occupation :

LMP :

EDD :

Any Specific Complaints :

Menstrual History:

Marital History:

Obstetric History :

Personal / Past History:

Family History:

General Examination

Height:

Weight :

Vitalsigns :-

PR :-

BP :-

CVS:

RS:

Obstetric Examination:

Investigations :

1. Blood 2. Urine

3. USG obstetrics:

4. Doppler – Uterine artery :

Presence of notch - Unilateral

- Bilateral

Absence of notch -

5. Development of HTD/ FGR :

6. Mode of delivery:

Vaginal

Caesarean

7. Gestational age at delivery

8. Perinatal outcome:

Birth weight

APGAR

NICU Admission

MASTER CHART